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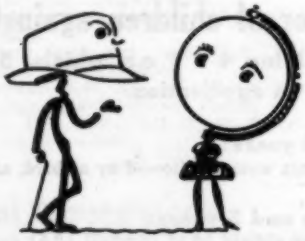

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EXPERIMENTAL MUMPS: TRANSMISSION OF THE DISEASE TO MONKEYS: ATTEMPTS TO PROPAGATE THE VIRUS IN DEVELOPING HENS' EGGS.

By CHARLES SWAN[1] and JESSICA MAWSON,[2]

From the Institute of Medical and Veterinary Science, Adelaide, South Australia.

THE present investigation was undertaken with the object of isolating the virus of mumps and of determining whether or not it could be propagated in developing hens' eggs. The relevant literature has been reviewed by van Rooyen and Rhodes[(1)] and Wesselhoeft.[(2)]

Material and Methods.

Saliva from 36 patients, collected according to the technique of Johnson and Goodpasture,[(3)] was obtained usually within twenty-four hours of the onset of parotitis, and always within forty-eight hours. In four cases not more than twelve hours elapsed between the onset of parotitis and the collection of saliva (see below). Twenty to forty cubic centimetres were obtained in one or one and a half hours, or larger quantities were obtained overnight. Sometimes we centrifuged the saliva at 2,000 revolutions per minute for thirty minutes to throw down the mucus; the supernatant fluid was then passed through a Berkefeld N filter previously "lined" with broth. Once, we substituted filtration of the saliva through moistened asbestos pulp in a Buchner funnel for the preliminary centrifuging. At first we inoculated saliva into monkeys within two hours of collection; later we froze the specimens at −10° C. until they were required. With few exceptions, the specimens obtained from two to five patients were pooled prior to inoculation (Table I).

[1] Working with the aid of a grant from the National Health and Medical Research Council.
[2] Working with the aid of grants from the Commissioners of Charitable Funds, Adelaide, and the University of Adelaide.

TABLE I.

Results of the Inoculation of Saliva from Human Cases of Mumps into Monkeys.

Number of Monkey.	Inoculum, and Time (in Days) Elapsing between Collection and Inoculation.	Routes of Inoculation, and Dosage. (Cubic Centimetres.)	Result (Including Day of Onset of Parotitis).
M929	Unfiltered saliva.	Right intraparotid (2·0)	?[1]
M930	Unfiltered saliva.	Right intraparotid (2·0)	?
M915	Unfiltered saliva.	Right intraparotid (2·0)	−[2]
M917	Unfiltered saliva.	Right intraparotid (2·0) Left intraparotid (2·0)	−
M938	Unfiltered saliva.	Right intraparotid (4·0) Left intraparotid (4·0)	−
M939	Unfiltered saliva.	Right intraparotid (4·0) Left intraparotid (4·0)	−
S129	Unfiltered saliva.	Right intraparotid (5·0) Left intraparotid (2·0)	−
M946 (*Macaca irus*)	Unfiltered saliva.	Nasal and oral spraying on three successive days.	−
M937	Unfiltered saliva.	Intratesticular (0·25) ..	−
S82	Filtered saliva (bacteria-free).	Intravascular (8·0) Intramuscular (20·0) Intracerebral (1·0)	−
S109	Filtered saliva (bacteria-free).	Right intraparotid (4·0) Left intraparotid (4·0) Intratesticular (0·5) Intramuscular (20·0)	−
S136 (*Macaca irus*).	Filtered saliva M.A.W.C. (bacteria-free) (21).	Right intraparotid (2·0) Intracerebral (1·0) Intramuscular (5·0)	+[3](10)
S142 (*Macaca irus*).	Unfiltered saliva M.A.W.C. (56).	Right intraparotid (2·0) Intramuscular (6·0)	+ (13)
S146 (*Macaca irus*).	Unfiltered saliva M.A.W.C. (70).	Left intraparotid (2·0) ..	+ (16)

[1] ? = Doubtful result.
[2] − = Negative result.
[3] + = Positive result.

We preserved the tissues (parotid gland and brain) of monkeys used in these experiments in 50% buffered glycerol solution (pH 7·0) at −10° C. Suspensions (5% to 10%) prepared by grinding with sterile washed sand and normal saline solution were centrifuged for five or ten minutes at 3,500 revolutions per minute; the supernatant

fluid was used as the inoculum. The dosage of tissue suspensions was one cubic centimetre given intracerebrally, three to ten cubic centimetres given intramuscularly, 0·25 cubic centimetre given into a testicle, and/or one to four cubic centimetres given by the intraparotid route. The dosage of cerebro-spinal fluid from patients suffering from clinical mumps meningo-encephalitis, when administered by the intraparotid route, was two cubic centimetres, given by the intracerebral, intramuscular and intravenous routes (0·5 to 1·0 cubic centimetre, 5·0 cubic centimetres, and 1·0 to 10 cubic centimetres respectively). Rabbits, guinea-pigs and mice respectively received intracerebrally 0·5 cubic centimetre, 0·25 cubic centimetre and 0·025 cubic centimetre.

In the egg culture experiments we used hens' eggs incubated for nine to twelve days, with intervals between each passage of four to seven days. Inoculations were given by the chorio-allantoic and intraamniotic routes according to the techniques of Burnet.[4][5] Chick embryos were also injected directly. The earlier steps in this method were similar to those of Burnet. Incisions were then made in the chorio-allantoic and amniotic membranes, and the portion of the embryo which presented itself was injected. After a number of passages (usually six), the eggs were tested for the presence of mumps virus by the inoculation of material into monkeys. After chorio-allantoic and intraembryonic inoculation, 10% to 20% suspensions of chorio-allantoic membranes and of embryos respectively were used. After intraamniotic inoculation, the amniotic and allantoic fluids were pooled prior to their injection. The dosage of the inocula in test monkeys was similar to that of the tissue suspensions.

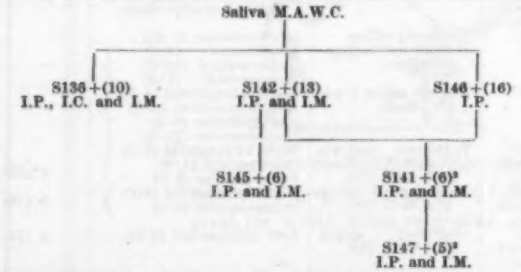
The monkeys used included 23 rhesus monkeys (*Macaca mulatta*) weighing 3·5 to 4·5 kilograms, 19 cynomolgus monkeys (*Macaca irus*), two pig-tails (*Macaca nemestrinus*), one of which weighed 4·6 kilograms, and one moor (*Macaca maurus*). The temperatures of all animals were recorded daily, and the animals were observed for up to one month from the time of inoculation. In a number of instances white blood cell counts were made at intervals of one or two days.

Experimental Procedure and Results.

Isolation in Monkeys of the Virus from Human Saliva and Transmission of the Disease in Series.

In all, fourteen attempts were made to transmit mumps to monkeys by the use of human saliva (see Table I). Only the last three attempts were successful. By injecting suspensions of the excised parotid glands of the animals thus affected into further monkeys, it was possible to transmit the disease in series (three passages; see Table II). In all cases the excised glands were proved to be sterile.

TABLE II.
Serial Transmission of Mumps in Monkeys from Saliva M.A.W.C.[1]



[1] The following abbreviations are used: "+" = a positive result; "I.P." = intraparotid inoculation; "I.C." = intracerebral inoculation; "I.M." = intramuscular inoculation. The numbers in parentheses indicate the incubation period of the parotitis, in days.
[2] S141 and S147 had been used previously as test animals in an attempt to detect the presence of virus in egg passage material, with negative results (see Table V).

For the most part the saliva was injected by means of a cannula passed along the parotid duct, according to the technique of Johnson and Goodpasture.[3] In some instances other or additional routes were used.

All three positive results were obtained from a specimen of saliva M.A.W.C., which comprised the pooled saliva collected from four patients within four to twelve hours of the onset of parotitis. A period of fourteen days elapsed between the collection of the samples from the first and last of the patients. From the end of this period, saliva M.A.W.C. had been kept frozen at -10° C. for 21, 56 and 70 days respectively prior to its inoculation into S136, S142 and S146; the incubation period in these animals was ten, thirteen and sixteen days respectively.

In contrast, the incubation period in the passage animals was five or six days. With this difference, the clinical picture in S136, S142 and S146 and in the monkeys used in passage of the virus was extremely uniform. The swelling of the parotid gland, which followed immediately upon the intraparotid inoculation, disappeared by the following day, and was not associated with pyrexia. Sometimes there was a mild rise in temperature of 1° or 2° F. on the day preceding the onset of parotitis; but the temperature fell to normal once swelling of the gland occurred. The parotitis was characterized by a swollen, tense, indurated gland with œdema of the overlying tissues (Figure I). Swelling usually reached its maximum on the second day, when œdema also involved the adjacent oral pouch. In most animals the parotid gland was then excised, but in monkey S145, which was not operated upon, the swelling and œdema took two further days to disappear. In no animal was the uninoculated gland affected.

FIGURE I.
Monkey S141; left-sided parotitis (seventh day after inoculation).

The symptoms in the foregoing animals were in marked contrast to those in monkeys injected by the parotid duct with negative results. In the latter, except when bacteria-free saliva was used, the swelling following inoculation persisted for two to four days and was associated with a leucocytosis and with a rise in temperature reaching 104° F. or 105° F. In only two instances (M929 and M930) was there any secondary swelling of the parotid gland comparable with that in the animals giving positive results. In these, the swelling following inoculation almost subsided, but reappeared on the sixth or seventh day, when it was accompanied by a rise in temperature to 104° F. Attempts were made to transmit the disease in series. Two monkeys (M921 and M920) were injected with a suspension of parotid gland from M929, and four animals (M916, M943, M922 and M944) with similar material from M930. The results were negative.

The initial pyrexia, and the fact that the swelling of the parotid gland following injection of unfiltered saliva

persisted for two to four days, were attributed to the presence of bacteria. It should be noted, however, that in the case of saliva M.A.W.C. there was no difference in the immediate symptoms which followed the injection of filtered (bacteria-free) or unfiltered samples. Probably the prolonged freezing of saliva M.A.W.C. had led to a decrease in the number and virulence of the bacteria.

Isolation in Monkeys of the Virus from the Cerebro-Spinal Fluid of a Human Patient with Mumps Meningo-Encephalitis.

By the use of specimens of the cerebro-spinal fluid of four human patients suffering from mumps meningo-encephalitis, six attempts were made to isolate the virus. Positive results were obtained from only one of the four patients; from this the disease was transmitted in series (see Tables III and IV).

TABLE III.

Results of the Inoculation of Cerebro-spinal Fluid from Human Patients with Mumps Meningo-encephalitis into Monkeys.

Number of Monkey.	Number of Case from which Cerebro-spinal Fluid was Obtained.	Routes of Inoculation and Dosage. (Cubic Centimetres.)	Result.
S118	I[1]	Intravenous (10·0)	+[2]
S124	I	Intracerebral (1·0)	+
S135	II	Intracerebral (0·5) } ..	−[3]
		Intravenous (1·0) }	
S138	III[1]	Intracerebral (0·6) }	−
		Right intraparotid (2·0) }	
S140	III	Right intraparotid (2·0) }	−
		Intramuscular (5·0) }	
S139	IV	Right intraparotid (2·0) }	−
		Intramuscular (1·0) }	

[1] In Case I rabbits, mice and a guinea-pig, and in Case III mice, were also inoculated with cerebro-spinal fluid, with negative results.
[2] + = positive result.
[3] − = negative result.

Monkey S118 was inoculated intravenously. On the previous day an attempt had been made to inject it with the same sample of cerebro-spinal fluid via the parotid ducts, but without success. It is possible, however, that a minute portion of the inoculum lodged near or in the orifices of the ducts. On the ninth day parotitis developed on the right side; it was not associated with pyrexia.

Monkey S124, which was injected intracerebrally, seemed well clinically, but on the twentieth day there was a slight rise in temperature, which reached 104° F. on the following day. Examination of the cerebro-spinal fluid on the twenty-fourth day revealed 950 white cells per cubic millimetre. There was no obvious parotitis. The animal was killed; later, histopathological examination revealed a chorio-meningo-encephalitis.

In addition to monkeys S118 and S124, a guinea-pig, rabbits and mice were also inoculated. The guinea-pig and mice remained well, but one rabbit became pyrexial on the thirteenth day. Four rabbits injected intracerebrally with a suspension of brain tissue from this animal remained free from symptoms. Histological examination of the brain of the rabbit originally inoculated revealed infection with *Toxoplasma cuniculi*.

Following the positive result in S118, an attempt was made to transmit the disease in series. The first attempt (S123) was unsuccessful. In this case the inoculum had been filtered through a Berkefeld N filter owing to contamination of the parotid gland of S118 with a few colonies of *Staphylococcus albus*.

After the parotid gland of S118 had remained in glycerol solution at −10° C. for twenty-four days, it was found by cultural methods to be sterile. Inoculation of a suspension of the gland into S129 led to swelling of the right parotid gland on the eleventh day. The positive result is of interest, in view of the fact that S129 had been injected about two months previously with saliva from mumps patients, with negative results.

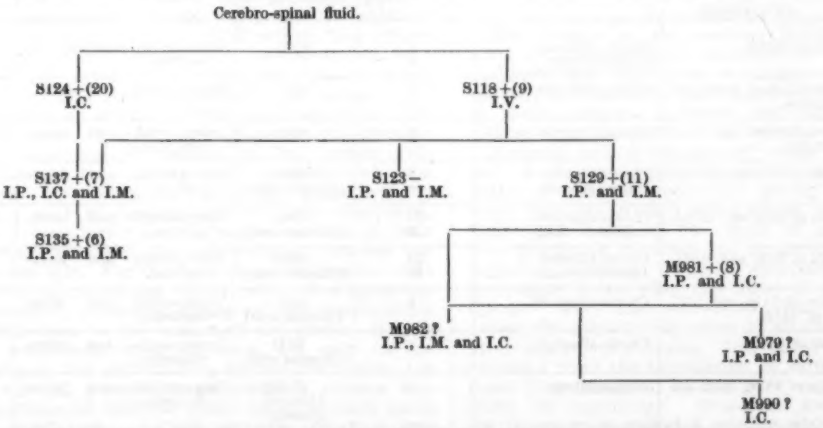
A suspension of parotid gland from S129 administered to M981 (*Macaca irus*) led to parotitis on the eighth day. Examination of the cerebro-spinal fluid revealed 250 cells per cubic millimetre, mostly mononuclear leucocytes. Despite the clinical condition of the animal, its temperature was normal.

The results of attempts at further passage of the virus must be looked upon as doubtful. In the case of M979 (*Macaca irus*), which was inoculated with the cerebro-spinal fluid of M981 combined with a suspension of parotid gland of the same animal, it was questionable on the ninth day whether the right parotid gland was slightly swollen. There were 112 cells per cubic millimetre in the cerebro-spinal fluid; the number had fallen to 26 by the following day. In M990 (*Macaca nemestrinus*) and M982, which were used in further attempts at passage, the maximum number of cells in the cerebro-spinal fluid were 85 and 387 per cubic millimetre on the seventh and twelfth days respectively. In neither monkey, however, did parotitis occur.

At a later stage of the investigation two successful passages of the virus were obtained in S137 (*Macaca irus*) and S135 (*Macaca irus*). The inoculum given to the former animal consisted of a suspension of brain tissue of S124 combined with a suspension of parotid gland of S118 (see Table IV).

TABLE IV.

Transmission of Mumps by Means of Cerebro-spinal Fluid from a Human Patient with Mumps Meningo-encephalitis.[1]


Cerebro-spinal fluid.
S124 +(20) I.C.
S118 +(9) I.V.
S137 +(7) I.P., I.C. and I.M.
S123 − I.P. and I.M.
S129 +(11) I.P. and I.M.
S135 +(6) I.P. and I.M.
M981 +(8) I.P. and I.C.
M982 ? I.P., I.M. and I.C.
M979 ? I.P. and I.C.
M990 ? I.C.

[1] The following abbreviations are used: "+" = a positive result; "−" = a negative result; "?" = a doubtful result; "I.P." = intra-parotid inoculation; "I.C." = intracerebral inoculation; "I.M." = intramuscular inoculation; "I.V." = intravenous inoculation. The numbers in parentheses indicate the incubation period of the parotitis or chorio-meningo-encephalitis (S124), in days.

Attempts to Grow the Virus of Mumps in Eggs.

Chorio-allantoic Passage.—At each passage the membranes were examined for the presence of macroscopic lesions. On several occasions small areas of opacity with ill-defined edges were seen; these may not have been due to non-specific causes. As a test of their specificity the passage material from the twelfth passages of attempts 4*b* and 4*c* (see Table V) were titrated (10^{0}, 10^{-1}, 10^{-2}) on membranes of twelve-day embryos. The eggs were opened on the third and fifth days, but although there were some lesions, their occurrence through the series was not consistent with the dilutions. (The adaptation of influenza virus to form lesions consistently on the chorio-allantoic membrane was accomplished by Burnet[3] only at about the fortieth egg passage; it is possible that the virus of mumps will prove as difficult to establish.)

Intraembryonic and Intraamniotic Passage.—At intervals during the series embryos were examined for macroscopic abnormalities (head, lungs, liver, muscles, skin *et cetera*). In addition, the turbidity of allantoic and amniotic fluids was observed. However, except in the sixth passage of attempt 7 (Table V), in which the amniotic fluid was unusually turbid, no abnormality was seen. Embryos from the thirteenth intraembryonic passages of attempts 4*b* and 4*c* were allowed to hatch; the chickens appeared normal, and when they were killed on the second day no abnormality was revealed.

Results.—Of the eight attempts which were made to propagate the virus, two (attempts 4*a* and 7) gave suggestive results (see Table V). In attempt 4*a*, monkey M983 (*Macaca irus*) was inoculated intracerebrally and in the right parotid gland with a bacteriologically sterile mixture of the sixth intraembryonic and fifth chorio-allantoic passages of a suspension of parotid glands of S129 and M981 in eggs. Prior to injection, the number of cells in the cerebro-spinal fluid was 40 per cubic millimetre (due to previous intracerebral inoculation); on the fourth, sixth and ninth days after inoculation it was 174, 132 and 290 respectively. On the ninth day the animal suddenly became very drowsy, its temperature was subnormal and convulsions occurred. Both of the parotid glands were swollen and the vulva was œdematous. One attempt at culture of the brain was without result; the other produced a growth of staphylococci. The result can be looked upon only as suggestive, as no attempt was made to transmit the disease to a second monkey. Moreover, bilateral parotitis developed, despite the fact that only one side was inoculated. Later (twenty-first and twenty-second) passages (attempts 4*b* and 4*c*) of this virus to monkeys M991 (*Macaca irus*) and M992 (*Macaca irus*) were unsuccessful. It was considered likely that the virus might grow more readily if it were injected by the intra-amniotic route and thus came into intimate contact with the embryo. Moreover, in case the virus grew very slowly, an interval of six or seven days was allowed to elapse between each passage. Material from the sixth intra-amniotic passage of parotid glands of S141, S142 and S146 (attempt 7) was injected into M1010 (*Macaca maurus*) by the right intraparotid and intramuscular routes; right-sided parotitis developed on the sixth day. A suspension of the gland (sterile on attempted culture) inoculated into S148 (*Macaca nemestrinus*) led to swelling of the parotid gland after a similar interval. In view of the fact that the parotitis had proved transmissible to a second monkey, this result was at first considered positive. However, when these monkeys were reinoculated by the intra-parotid route with the original virus (derived from S141, S142 and S146), M1010 developed parotitis on the sixth day, while S148 remained well. The reason for this result is by no means clear. A further attempt (attempt 8) at propagation of the virus was made by means of material derived from S141, S142, S146 and S147. The test animal, S149, remained free from symptoms. However, as no parotitis developed on reinoculation with the original virus, either we were dealing with a naturally immune monkey, or the first inoculation had led to a subclinical infection rendering the animal immune. Certain of the negative results (attempts 1, 2 and 3) were not unexpected, as it was never demonstrated that the original inoculum of the eggs contained mumps virus. In attempts 5 and 6, however, despite the fact that the original inoculum of the eggs contained the virus, the test monkeys (S141 and S147) remained well. These results must be looked upon as certainly negative, as on reinoculation with mumps virus, both S141 and S147 developed parotitis (see Table II).

Immunity Tests.

At the conclusion of the foregoing experiments, five of the monkeys were tested for immunity. In the case of S139 and S145 the parotid gland previously inoculated was reinjected. As the parotid gland originally

TABLE V.

Attempts to Grow the Virus of Mumps in Developing Hens' Eggs.

Number of Attempt.	Source of Material with which Eggs were Originally Inoculated.	Routes of Inoculation in Eggs.	Number of Passages in Eggs.	Number of Monkey Used to Detect Virus in Egg Material.	Routes of Inoculation in Monkeys.	Result (including Incubation Period in Days).
1	Parotid gland of M930.	Chorio-allantoic. Intraamniotic ..	6 6	M945	Intraparotid, nasal and oral spraying.	—[1]
2	Filtered (bacteria-free) saliva of mumps patients.	Chorio-allantoic ..	6	S94	Intraparotid.	—
3	Filtered (bacteria-free) saliva of mumps patients.	Intraembryonic ..	6	S96	Intraparotid and intramuscular.	—
4*a*	Parotid glands of S129 and M981.	Chorio-allantoic .. Intraembryonic ..	5 6	M983 (*Macaca irus*)	Intraparotid and intracerebral.	?[2] (9)
4*b*	Parotid glands of S129 and M981.	Chorio-allantoic .. Intraembryonic ..	21 22	M991 (*Macaca irus*)	Intraparotid and intracerebral.	—
4*c*	Parotid glands of S129 and M981.	Chorio-allantoic .. Intraembryonic ..	21 22	M992 (*Macaca irus*)	Intraparotid and intracerebral.	—
5	Parotid glands of S118 and S137 and brain of S124.	Chorio-allantoic ..	6	S141 (*Macaca irus*)	Intraparotid and intramuscular.	—
6	Parotid gland of S142.	Chorio-allantoic ..	6	S147 (*Macaca irus*)	Intraparotid and intramuscular.	—
7	Parotid glands of S141, S142 and S146.	Intraamniotic ..	6	M1010 (*Macaca maurus*).	Intraparotid and intramuscular.	? (6)
8	Parotid glands of S141, S142, S146, S147.	Intraamniotic ..	8	S149	Intraparotid and intramuscular.	—

[1] —=no result.

[2] ?=doubtful result.

inoculated had been excised from monkeys S136, S141 and S147, the remaining gland was injected. In addition, all of the animals, with the exception of S136, were inoculated intramuscularly. S136, S141, S145 and S147 had had clinical mumps, S139 had not; all five animals remained free from symptoms. A point of interest was the immunity of monkey S139. At one stage an attempt was made to inoculate the animal with saliva M.A.W.C. via the parotid ducts. Although the attempt was unsuccessful, a small portion of the inoculum lodged in the mouth. Clinically, however, the animal remained well, and on this account and owing to a severe shortage of experimental animals, was used later to test the cerebro-spinal fluid of the patient in Case IV (see Table III), with negative results.

As saliva M.A.W.C. was known to contain mumps virus, it seems fair to assume that the contamination of the oral cavity with the infected saliva led to a subclinical infection resulting in immunity. It may be mentioned that Johnson and Goodpasture[3] found in the majority of cases that nasal and oral spraying of monkeys with mumps virus led to immunity without the development of clinically recognizable infection.

Attempts to Transmit the Virus of Mumps to Dogs, Rabbits and Mice.

Two dogs which were inoculated intramuscularly and by means of nasal and oral spraying with mumps virus remained free from symptoms. Similarly, no result followed the intracerebral injection of two rabbits. It was hoped that the virus might be adapted to mice by rapid passage from animal to animal without waiting for the development of symptoms. Accordingly five young (five weeks old) mice were inoculated intracerebrally with mumps virus. Two of the mice were killed on the sixth day and a suspension of their brains was injected into further mice. Four passages were carried out, three mice of each passage being retained for observation. None of the animals developed symptoms.

Pathological Examination.

Parotid Glands.

Macroscopic Findings.—Reflection of the œdematous overlying tissues of the parotid glands examined revealed a tense, indurated, œdematous, congested gland about three times the normal size, and with its surface covered with petechial hæmorrhages. As similar naked-eye appearances were encountered in monkeys M929 and M930, which were inoculated with unfiltered saliva with doubtful results, it cannot be claimed that the above appearances are necessarily characteristic of mumps.

Histological Examination.—The glands were fixed in Zenker-acetic solution and embedded in paraffin. Sections 4·0μ to 6·0μ in thickness were stained with hæmatoxylin and eosin, phloxin-methylene blue and Masson's trichomic stain. In monkeys in which inoculation had produced positive results the pathological process was fairly uniform, but it varied in intensity; it was focal rather than diffuse in distribution. Severe lesions were characterized by a very inflamed and œdematous gland, its lobules much infiltrated with lymphocytes, plasma cells and mononuclear cells, together with occasional eosinophile leucocytes. The majority of its acini showed either degenerative changes or necrosis. Degenerative changes in the acinar cells included swelling of the cells, sometimes associated with separation from their basement membrane, granularity or vacuolation (or both in combination) of their cytoplasm, and shrinkage, irregularity and hyperchromatism of their nuclei. In many instances the degenerative changes had progressed to necrosis. For the most part whole acini were affected rather than individual acinar cells. The epithelial cells lining the ducts remained unaffected, but eosinophilic debris was often seen in the lumina of the ducts. Minute hæmorrhages were sometimes observed, not only in the lobules, but also in the surrounding connective tissue. In less severely affected glands the pathological process was confined to portions of lobules, while in mild cases some lobules were unaffected. Actual necrosis occurred less frequently. The inoculated parotid glands of all the monkeys injected either with saliva M.A.W.C. or with material from its subsequent passages were severely involved, but animals S142, S141 and S147 were more affected than S136 or S146 (see Table II). In contrast, the pathological process in the monkeys inoculated with mumps virus derived originally from cerebro-spinal fluid (Table IV) was less severe. While S118, S129 and S137 were moderately affected, M981 was affected only mildly. The parotid gland of monkey M1010 (attempt 7, Table V), which gave a suggestive result with egg material, was as severely involved as that of the monkeys inoculated with the virus derived originally from saliva. On the other hand, the parotid gland of monkey M983 (attempt 4*a*) was only mildly involved. Histological examination of the inoculated parotid glands of M929 and M930, which were injected with saliva with doubtful results, revealed a severe inflammation comparable in intensity with that described in the positive cases. There were, however, certain differences; in monkey M929, for instance, a number of giant cells of the foreign body type were detected, while in M930 proliferating fibroblasts and foci of polymorphonuclear leucocytes were seen. Polymorphonuclear leucocytes in the "positive" cases, however, were conspicuous by their absence.

Central Nervous System.

Macroscopic Findings.—Apart from the presence of mild congestion, the brains were not remarkable.

Histological Examination.—The brains were fixed in formol-saline solution and embedded in celloidin. Sections 10μ to 15μ in thickness were stained by routine neurohistological methods. In the case of S124 (Tables III and IV), examination of sections disclosed a moderate to severe pia-arachnoid infiltration with lymphocytes and adventitial cells. Rarely, a few polymorphonuclear leucocytes were seen in the infiltrate. The infiltration was patchy, and was much more obvious basally and in the depths of the sulci. It was especially severe in the region of the brain stem and cerebellum, and it was well in evidence in the spinal cord. The brain was congested, particularly in the tissues immediately adjacent to the ventricles (for example, the hypothalamus). Severe perivascular infiltration of the vessels adjoining the lateral, third and fourth ventricles was present, together with diffuse and focal infiltration of the ependyma and sub-ependymal tissues. There were pronounced hæmorrhages and severe infiltration (mainly lymphocytic) of the chorioid plexuses; these changes could not be seen in all sections, nor in all of the plexus in any section affected. In the case of M983 (Table V) there was a mild patchy meningitis composed of lymphocytes and adventitial cells together with considerable numbers of polymorphonuclear leucocytes. After long search two vessels were found in the cortex with mild perivascular cuffing. Apart from a minute hæmorrhage in the pons, no other pathological change was found.

Discussion.

The results of the present investigation confirm the work of Johnson and Goodpasture[3] and of Findlay and Clarke[4] that the virus of mumps can be isolated from the saliva of patients suffering from the disease. Contrary to their findings, however, we experienced considerable difficulty in isolation of the virus.

Johnson and Goodpasture[3] were successful with four out of six specimens of saliva collected from mumps patients. Of the specimens giving positive results, two were collected within twenty-four hours and two within forty-eight hours of the onset of parotitis; in contrast, two specimens obtained on or about the third day gave negative results. Using a specimen of saliva collected forty-eight hours after the onset of parotitis, Findlay and Clarke[4] were also successful; but they were unable to detect the presence of virus in another specimen collected after seventy-two hours. Our three positive results were obtained from the inoculation of saliva M.A.W.C., collected from four patients within four to twelve hours of the onset of parotitis. However, despite the fact that specimens were collected usually within twenty-four hours, with two doubtful exceptions the results of eleven earlier attempts made with saliva from 32 patients were unsuccessful; eight of these attempts at inoculation were

made by the intraparotid route. The reasons for our difficulty in isolation of the virus are by no means clear. It is possible, of course, that the virus disappears rapidly from the saliva after the onset of parotitis, and that to detect it after twelve hours, as has been done by the earlier investigators, is exceptional.

On the other hand, our results may be due to other factors in the experimental conditions. The possibility of the presence of a virus-inactivating agent in saliva, similar to that described by Burnet, Lush and Jackson[9] in human nasal secretion, was considered. We believe, however, that we have eliminated this factor by packing the specimens of saliva on ice during transport and by keeping them frozen at -10° C. until required. Burnet, Jackson and Robertson[10] showed that cynomolgus monkeys (*Macaca irus*) were more susceptible to poliomyelitis virus than rhesus monkeys (*Macaca mulatta*). Perhaps the former species is also more susceptible to mumps virus, but until further work is done no opinion can be expressed on this question. Earlier workers had no difficulty in infecting rhesus monkeys (*Macaca mulatta*). Johnson and Goodpasture[7] were unable during the summer months to induce experimental mumps in monkeys with any degree of regularity. They assumed that in some obscure way the hot weather, associated with factors over which they had no control, was responsible. It seems doubtful whether climatic conditions played any part in our results, because, although the major portion of our work was done during the summer months, we obtained positive as well as negative results during that period. Moreover, once the virus had been isolated, it was transmitted through further passages without difficulty. A further possibility is that in some cases we may have been dealing with naturally immune monkeys.

Two points of interest emerge from the study of saliva M.A.W.C. The first was the survival of the virus in the saliva for at least seventy days when the material was frozen at -10° C. The second was the gradual increase of the incubation period (from ten to sixteen days) of the parotitis resulting from its inoculation, when the saliva was kept frozen for increasing periods of time (twenty-one to seventy days). This result suggests that storage led to a gradual decrease of virulence of the virus. That such loss of virulence was only temporary was shown by the fact that on further passage the incubation period fell to five or six days (see Table II). The longest incubation period of sixteen days (monkey S146) closely approached the average incubation period in man of eighteen days.

The doubtful results in monkeys M929 and M930 (Table I) are of interest, in that they closely resemble those of Levaditi, Martin, Bonnefoi and Schoen.[11] Their attempts at transmission of the disease through further passage, like ours when using material from M929 and M930, were unsuccessful.

In general, the histological findings in the parotid glands were similar to those described by Johnson and Goodpasture[3][12] and by Findlay and Clarke.[8] The pathological process which resulted from inoculation of mumps virus derived originally from cerebro-spinal fluid was less severe than that which followed the injection of virus isolated from saliva; this fact suggested that the former was a less virulent strain.

Nevertheless, the virus derived originally from the cerebro-spinal fluid was capable of inducing a severe chorio-meningo-encephalitis when inoculated intracerebrally (monkey S124; Tables III and IV). The pathological findings in this animal bore a pronounced resemblance to those described by Johnson and Goodpasture[7] in a monkey (M96) which they injected intracerebrally with a suspension of parotid gland containing mumps virus. There is less resemblance to the histopathological picture described by Gordon,[13] who inoculated filtered saliva from mumps patients intracerebrally into monkeys. Moreover, the incubation period of monkey S124 was twenty days, while Gordon's successful cases terminated fatally on the fourth day.

In our attempts to cultivate the virus of mumps in developing hens' eggs, we obtained two suggestive but not necessarily positive results. However, even if it is eventually shown that the virus can be propagated with regularity in eggs, it will still be necessary to use monkeys to detect the growth of virus, unless regularly recurring specific lesions develop on the chorio-allantoic membrane. Our main object was to find a simple and inexpensive method of studying the virus, perhaps leading to the production of a vaccine. Unfortunately, owing to lack of further monkeys brought about by the war, we are unable to repeat these experiments on a larger scale.

The results of the immunity tests confirm those of Johnson and Goodpasture.[3][7]

Like Findlay and Clark,[8] we attempted unsuccessfully to infect rabbits, a guinea-pig and mice with mumps virus. In view of the fact that Busquet and Laveran[14] noted that a dog belonging to a mumps patient developed parotitis, we tried unsuccessfully to infect dogs. Unfortunately, we were unable, despite several attempts, to inoculate the virus by the intraparotid route, and had to be content with nasal and oral spraying together with intramuscular injection. In view of the possibility that dogs might behave like monkeys—that is, develop clinically recognizable parotitis only when the injection is given directly into the parotid gland—we cannot deny the possibility that dogs may be susceptible to the disease.

Summary.

The virus of mumps has been isolated from the pooled saliva of four patients suffering from mumps, and from the cerebro-spinal fluid of a patient suffering from mumps meningo-encephalitis. Each of the two strains of virus has been transmitted through three passages in monkeys. The symptomatology and pathology of the experimental disease have been discussed.

Two of eight attempts to cultivate the virus of mumps in developing hens' eggs gave suggestive, but not necessarily positive, results.

Rabbits, mice, guinea-pig and dogs remained well when inoculated with mumps virus by various routes.

Acknowledgements.

Our thanks are due to Professor E. Weston Hurst for helpful criticism and advice, and to Squadron-Leader F. R. Wicks, of the Royal Australian Air Force, and to the Superintendent and Staff of the Infectious Diseases Hospital, Northfield, for assistance in obtaining samples of saliva.

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CONGENITAL ABNORMALITIES, TERATOLOGY AND EMBRYOLOGY: SOME EVIDENCE OF PRIMITIVE MAN'S KNOWLEDGE AS EXPRESSED IN ART AND LORE IN OCEANIA.

By ISADORE BRODSKY,
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THERE is evidence that natives of islands to the north east and north of Australia, as well of the continent itself, have had access to normal and abnormal embryological material, and have been impressed to such a degree as to cause it to be embodied, purposely or by chance, in legend and art. My attention was first drawn to the possibility through a mistaken idea that the familiar Maori tiki was intended to represent wry neck in an infant; the idea occurred to me about the time that I was working on congenital abnormalities.[1] Since that time I have made several visits to islands in the region indicated, and though only superficial contacts and observations have been permitted, I have been able to collect shreds of evidence from anthropologists and from traders, as well as museum specimens and references in anthropological literature. The evidence, tattered as it is, seems worthy of presentation, not only to afford a medical basis for a section of native art and lore, but also to provide an extra-interpretative link in the incomplete chain of knowledge concerning the cultural side of native life.

The map (Figure I) shows the distribution of the range of the materials, and emphasizes the present risk to which legends, specimens and the natives themselves are being exposed, under conditions of warfare in the Pacific.

FIGURE I.

Carvings, Drawings, Legends.

It is convenient to separate the material into three groups: carvings, drawings and legends, distributed as follows:

The Carvings:
- (i) New Ireland (two specimens).
- (ii) Papua.
- (iii) Solomon Islands.
- (iv) New Zealand.

The Drawings:
- (i) Dutch New Guinea.
- (ii) Queensland.
- (iii) New South Wales.

The Legends:
- (i) Samoa (two legends).
- (ii) Papua.

[1] Work done with the aid of a grant from the National Health and Medical Research Council in 1938.

Carvings.

The first carving from New Ireland is in the Australian Museum Collection; it has no number, and it was purchased from Parkinson and Farrell in 1885 (Figure II). The carving is of chalk and represents a double-headed human figure, of unindicated sex, in a sitting position. The sitting height is twenty inches, and the general proportions are those of a single figure. A mass which projects horizontally from the mid-line slightly below shoulder level, is held or supported by the two hands.

The second carving from Gardner's Island, New Ireland, is in the Australian Museum Collection, and has the number E 597. It was purchased from Captain Farrell in 1887 (Figure III). This wooden carving is about three feet six inches long and it has been coloured with red, black and white. Essentially, the carving shows the birth of a child, with the lower part of the body and the lower limbs not yet born.

FIGURE II.

FIGURE III.

The carving from Fife Bay, Papua, is in the Australian Museum Collection, and has the number E 27502. It was collected by Captain W. H. Steele, of the London Missionary Society's steamer *John Williams*, and was presented by Mrs. S. A. Steele in 1923 (Figures IV and V). The carving has been made in wood, the subject being a human. The length is eight inches. The attitude of the figure, especially from the oblique antero-lateral view, the facial expression and the deformed right wrist, are the interesting features.

The carving from Ugi (Uji) Island, Solomon Islands, is in the Australian Museum Collection and has the number B 428. It was purchased from Mr. J. Stephens in 1884 (Figure VI). This is a wooden carving of compact human male figures conjoined back to back from the occipital to the sacral regions. The carving stands fifteen inches high.

The carving from New Zealand is in the Australian Museum Collection and has no number. It was transferred from the Mitchell Library (Figure VII). The *hei tiki* has been carved from greenstone, and is four inches long.

Drawings.

The drawing from Dutch New Guinea was collected by De Clercq and Schmeltz[1] (Figure VIII). The drawing, made on a door, shows a human female figure in a squatting position. Within her body lies a male fœtus enclosed in a sac.

FIGURE IV.

FIGURE VI.

FIGURE V.

FIGURE VII.

The drawing from Stanley Island, Princess Charlotte Bay, Queensland, was collected by Hale and Tindale[2] (Figure IX). A rock drawing shows a human female figure, from which a cord passes from the vagina to be attached to the umbilical region of a smaller figure. The larger of the two is about six inches long.

The rock drawing or engraving near Berowra Waters, New South Wales (Figure X), shows a double-headed human male figure, nine feet six inches long. The artist has allotted six fingers to the right hand and four to the left hand.

Legends.

The two legends from Samoa, collected by Brown,[3] are as follows:

1. There was a woman called Faga; she brought forth a daughter whose name was Papa. Her vagina was imperforate.

1 2 3 4

FIGURE VIII.

2. Sinalolofutu became with child and she brought forth twin girls, but they were not separated, but were joined together by their backs. Their names were Ulu and Ona . . . After many years—the years were not known—the girls had grown up . . . the girls were startled in their sleep, and rushed from the house, each one by a separate door. The door post separated their bodies, so that they were parted asunder . . .

The legend from Purari Delta, Papua, collected by Williams,[4] is as follows:

More than one tale is told regarding the manner of Iko's birth. But this much is certain, that he was the son of a cassowary, and the foster son of two twin women, who were joined fast together, flesh and bone, by the buttocks . . . the two twin women, Apiko and Novido . . . of a certainty they were queer creatures joined back to back, so that when they would walk, the one must go forwards and the other backwards . . . while yet a boy, Iko saw how awkwardly the two women . . . were joined together; so while they slept at night, he took a bamboo and prised a sliver off it, with an edge sharper than any knife, and so neatly cut away the one from the other, that both slept soundly the while, feeling no pain, and in the morning got to their feet, two separate women . . .

FIGURE IX.

Interpretation of Material.

Now that the facts have been presented—and interesting facts they are—the next step is to cross indefinite boundaries into the province of interpretation. From the standpoint of the sciences of teratology, congenital abnormalities and embryology, an examination of the primitive material permits an attempt to relate and compare that material with well-known counterparts in the sciences named.

To deal with the specimens in order, the carving from New Ireland corresponds exactly with a *dicephalus dibrachius* monster, of which many specimens have been seen and described. Possibly the median horizontal projection, which the lateral arms are supporting, may represent fused deformed arms, in which event the carving represents a *dicephalus tribrachius* monster.

Obstetrically, the Gardner's Island figure requires no interpretation. The Fife Bay carving arouses interest for more than one reason. The right hand shows the condition of club hand, which may be either congenital or acquired. I regard it as an example of the congenital type because of coincidental abnormalities—namely, short right humerus (as compared with the left), and idiotic expression and stance, which with the protruding tongue may be taken as collateral evidence of cretinism, itself an abnormality of the thyreoid gland.

The wooden conjoined carving from Ugi (Uji) Island at once suggests Siamese twins of the pygopagus type, with additional union of the bodies and heads. There is also a suggestion of achondroplasia—the head is relatively large, the arms are short and the thighs are short and muscular.

Earlier, I drew attention to my misconception about the *hei tiki*, which allows an impression of wry neck. The impression is heightened by the asymmetry of the head, which assists in the distinguishing of the congenital type of wry neck from the acquired type. Elsdon Best[5] holds the opinion that the tiki represents a fœtus cramped *in utero*; but Skinner[6] finds that he cannot accept Best's interpretation. There appears to be agreement, however, on a relationship of the tiki to Hina-te-iwaiwa, the goddess who presides over childbirth.

The drawings from Dutch New Guinea and Princess Charlotte Bay demand no special interpretation, because they clearly convey their embryological or obstetric meaning. With equal clearness the Australian aboriginal has engraved a dicephalus monster.

Details of the Samoan and Papuan legends leave no doubts that the monsters are pygopagi, in each case the conjoined elements being true to scientific pattern—in their sex, as a noteworthy particular. In regard to the Samoan legends there is an extra point to be brought out, which is not shown in my extracted version; the woman with the imperforate vagina, according to the legend, eventually becomes the mother of the pygopagi—an example of congenital abnormality in two generations of the one family.

Is it possible that natives have seen these monsters, which occur so infrequently even in denser populations? None can say. Yet ways suggest themselves of establishing an answer. Firstly, such monsters are well known to science. Secondly and broadly, monsters are not restricted to species, race or zone. And then, there is the evidence of accurate description and representation.

The translated version of the legends about the pygopagi—two beings of the same sex joined fast together, flesh and bone, by the buttocks—could apply readily to the famous Blazek sisters, Violet-Daisy Hilton, and others listed and described by Newman.[7] Haply united, too, are the male figures in the Solomon Island specimen.

FIGURE X.

In support of the specimen and of the legend concerning minor abnormality, many authentic instances of congenital anomalies have been recorded. Albinism is the commonest. Williams in "The Papuan Villager" mentions a boy from Goaribari village with six digits on each hand and foot—"he is very proud of his extra fingers"; he also mentions another boy from the Chirima Valley, with an extra toe on each foot. Guppy[8] observed one example of hare-lip in association with hypertrichosis in a man of Simbo, and he also saw, at Ugi (Uji), a man who had six perfect toes on the right foot. Taplin[9] records of the Narrinyeri that all deformed children were killed as soon as they were born, while Gason[10] says the same about the Dieyerie, "in fear of them becoming a burden to the tribe".

In regard to the embryological side, the artistic evidence demands greater tribute than the implied knowledge of developmental processes. While it is improbable that males were allowed to witness confinements, it is manifest that the artists were familiar with some aspects of the process. For example, the Dutch New Guinea specimen shows the male fœtus *in utero*, with the fœtal membranes in position. Possibly this drawing was a reconstruction from circumstantial evidence, for it is to be noted that the unborn fœtus bears only a portion of the umbilical cord.

The Artist's Motive.

The final consideration concerns itself with the motive of the artist; but I shall not mention belief, magic and myth, though they may be pertinent to the discussion. Whim may have played a part in the production of these carvings and drawings; but the detail, in the light of our knowledge, is too faithful for that. The appeal of the grotesque, though it must be very strong, may be a positive factor, and to a lesser extent the desire to caricature must also find a place in the art. Imitation of preexisting patterns explains their presence, but not their origin, while it is unreasonable to bring forward the suggestion of spontaneous creative art as the basis.

In support of the viewpoint I have suggested, N. B. Tindale, in a personal communication, writes: "I am sure that the occurrence of malformations, etc., in humans does give rise to stories and has an effect on mythology . . ." Primitive man is a remarkably keen observer, and therefore I think it just to assess the specimens from a medical standpoint, and as examples of realism in art.

Acknowledgements.

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Reviews.

ANTE-NATAL AND POST-NATAL CARE.

THE fourth edition of "Antenatal and Postnatal Care", by F. J. Browne, has been published.[1] In spite of the fact that the book has been revised throughout, and much new matter added, the size of the volume has by drastic pruning been reduced by thirty-two pages. The bibliography is now placed at the end of each chapter instead of at the end of the book, and this is a decided advantage.

New matter has been added chiefly in the chapters on unsuccessful pregnancy, vomiting of pregnancy, and the toxæmias of late pregnancy, while there has been a general revision throughout the book. The methods of preventing and treating stress incontinence following childbirth by physical therapy have been discussed. Greater emphasis has been laid on radiological methods in obstetric diagnosis and prognosis.

Unsuccessful pregnancy is discussed under four headings: (*a*) abnormalities in the genital organs of the mother; (*b*) general disorders of the mother; (*c*) defects in the male parent; (*d*) idiopathic group. Each of these sections is well written, and the influence of many factors, varying from fibroids to deficiency of vitamins *E* and *C*, and altered hormone deficiency including gonadotropic hormones, œstrin and progestin, is judicially considered. The author's conclusions are as follows: "It seems, therefore, that a moderate and fairly uniform degree of success follows the use of a large variety of apparently unrelated therapeutic measures in idiopathic habitual abortion, and it is not surprising if doubts are beginning to arise in the minds of some observers regarding the real value of any of them."

He quotes a series of eighteen cases of habitual abortion reported by Dr. Muriel Boycott, in which all patients had at least two, and most of them three spontaneous abortions, and none having had a child. Eight were treated by progestin, all successfully; three were treated by vitamin *E*, one being in hospital for five months, all successfully. Seven patients were controls; all were untreated, two were kept in hospital, and five were treated as out-patients. All seven patients gave birth to a live child at term.

The author concludes that many more such controlled series are required before a final judgement can be passed on the value in idiopathic habitual abortion of any of the methods of treatment described by him.

In the author's review of the causes of vomiting of pregnancy, mention is made of the claim by Schoeneck and others, that the urine in hyperemesis contains excess of gonadotropic hormone (prolan A and B). Possibly the excessive excretion of hormone is an indication of abnormal swelling of the anterior lobe, causing a sickness of cerebral origin. Kemp recently put forward a view that vomiting of pregnancy is due to a temporary insufficiency of the maternal adrenal cortex. In pregnancy there is a greater need for cortin (the secretion of the adrenal cortex), and therefore an insufficiency might arise until such time as the adrenal cortex adequately hypertrophies about the end of the third month. To be of value, ten to twenty cubic centimetres must be given daily by intramuscular injection.

From the pathological viewpoint, the author points out that lesions resembling Wernicke's encephalopathy have been recently described by Campbell and Biggart in the brains of two patients who died of *hyperemesis gravidarum* and in two cases described by Sheehan. These lesions may be caused by a toxin originating in the failure of the liver to detoxicate metabolic products, or some consider they are due to deficiency of vitamin B_1.

In the toxæmias of late pregnancy, a fuller account has been given of endocrine factors as they bear on ætiology, and the lately published observations of the author and Gladys H. Dodds on pregnancy in the patient with chronic hypertension have been included. This chapter is one of the best in the book.

We have strongly recommended previous editions of this book to all who practise obstetrics, and the present edition only serves to bring the subject matter more up to date. Those familiar with earlier editions will no doubt find this latest one very useful, and to those who are not yet familiar with it, we recommend it as a work which will give them great help in dealing with all those problems which sooner or later confront everyone who practises obstetrics.

[1] "Antenatal and Postnatal Care", by Francis J. Browne, M.D. (Aberdeen), D.Sc., F.R.C.S. (Edinburgh), F.R.C.O.G.; Fourth Edition; 1942. London: J. and A. Churchill Limited. Demy 8vo, pp. 616, with 84 illustrations. Price: 24s. net.

WAR INJURIES OF THE CHEST.

"WAR INJURIES OF THE CHEST", by H. Morriston Davies and Robert Cope, although only a small book of 128 pages, is full of practical and vital information.[1] It should be read and reread by all medical officers who may possibly be called upon to treat this type of injury either in forward areas or at base hospitals.

The authors have the happy knack of embodying basic physiological principles along with the details of treatment, as is exemplified in their frequent references to the dangers of anoxia which are brought clearly and forcibly before the reader.

The importance of the correct and adequate administration of oxygen is stressed and the dangers of nitrous oxide and chloroform as anæsthetic agents in such cases are indicated.

The short mention of "blast lung" epitomizes current opinion in a clear fashion. The necessity for care and gentleness in the handling of patients affected by this condition is emphasized and it is pointed out that an intelligent inspection often yields more information in the initial examination than does the laying on of hands.

The chapter on hæmothorax is first class and the necessity for early and repeated aspiration stands out in very clear fashion.

In the treatment of infected hæmothorax attention is drawn to the correct length and placing of the drainage tube and to the advisability of water-sealed drainage, but the very efficient and time-saving method of negative pressure drainage is not mentioned. Taken as a whole the main principles guiding correct treatment of penetrating and open wounds of the chest are given clearly and concisely and the illustrations used all have some practical value.

[1] "War Injuries of the Chest", edited by H. Morriston Davies and Robert Cope; 1942. Edinburgh: E. and S. Livingstone. 7¼" × 5", pp. 139, with illustrations. Price: 6s. net.

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THE PROPHYLAXIS OF ACUTE RECURRENT RHEUMATISM: A NOTABLE ADVANCE.

In a paper on rheumatic heart disease in this journal on August 3, 1940, H. Boyd Graham gave some extracts from the "Harveian Lectures" published in *The Lancet* in 1889. Here it was stated that the whole interest and importance of rheumatism in children centred in the cardiac inflammation and its results. The one great aim and object of treatment was stated to be to minimize this danger as much as possible, and details were given of the way in which a watch was to be kept and early symptoms were to be treated. Graham later on pointed out that most children who are going to do so, show evidence of permanent damage to the heart within two years of the onset of illness. It is perhaps well to call to mind the fact that a great deal of work has been done on the causation of acute rheumatism. The environmental conditions of patients have been studied and many workers have sought to isolate a microorganism that can be held responsible. That most of the evidence points to a streptococcus as being the *fons et origo mali* is well known, but it cannot be claimed that its culpability has been established beyond doubt; a virus has been suggested on reasonable grounds as being at least partly responsible. (See THE MEDICAL JOURNAL OF AUSTRALIA, November 13, 1937, page 885.) From the present state of our knowledge it follows that any prophylactic measures that are undertaken must be purely empirical. Were we able to bring up every child in the community in such healthy surroundings that he would acquire what has been aptly called rude health (the elect among the medical politicians talk of "positive health") he would probably be able to withstand the onslaught of microorganisms carrying rheumatic infection. As it is, children and adults are stricken by the infection known as acute rheumatism and the ultimate aim of treatment, as already stated, is to minimize the effects of the disease, particularly on the heart. These effects are not immediately evident, and acute rheumatism is a disease characterized by relapse or recrudescence. In the present state of knowledge, the views on the streptococcal nature of the infection being what they are, it is not surprising that drugs of the sulphonamide series have been put to the test in both treatment and prophylaxis—if the streptococcus was responsible, it was argued, the sulphonamide drugs should cut short the acute stage of the disease, and, taken in correct doses and at suitable intervals, should prevent the recurrences that produce such permanent damage in the cardiac and other tissues. The results obtained are interesting and somewhat unexpected. Stated shortly, it has been found that, given in the acute stages, the sulphonamides are normally ineffective and may be harmful; given prophylactically, after the subsidence of acute symptoms, they are effective in preventing recurrences. A. F. Coburn and L. V. Moore found that the administration of sulphanilamide after the onset of streptococcal throat infections, did not prevent rheumatic recrudescences.[1] In the "Seventh Rheumatism Review", published in 1941,[2] reference is made to papers which "confirm previous reports" that sulphanilamide is not beneficial, but may be actually harmful in the treatment of active rheumatic fever, or when given in the silent period after the onset of a precipitating sore throat. One of these papers is an article by W. T. Ritchie on acute rheumatic carditis in which he stated that sulphanilamide might aggravate arthritic pains and cause a rise in temperature.[3] Although they do not directly concern acute rheumatism, reference may be made to experiments by Coburn and Moore, reported in the abstract already mentioned, in which sulphanilamide was given to guinea-pigs before and after the induction of streptococcal abscesses; in these circumstances the sulphanilamide failed to sterilize the lesions, but when it was used prophylactically the drug prevented the occurrence of spontaneous infection.

The first detailed reference in this journal to the prophylactic use of sulphonamide drugs in acute rheumatism was made in June, 1941, in a discussion on the "Seventh Rheumatism Review". The first work mentioned was that by A. F. Coburg and L. V. Moore. (In error the name of R. H. Pauli, who had done some other work with Coburg on streptococci and rheumatism, was used.) These workers gave daily doses of two to three grammes for four to eight months to 80 children who were in the inactive or quiescent stage of acute rheumatism, and 79 of them escaped further signs of rheumatic activity. The next authors mentioned were Caroline B. Thomas and R. France. Some details of a first publication of theirs were given, but it was noted that they had, with F. Reichsman as a third member of the team, just published another article,[4] and it is to this that reference will now be made. Sulphanilamide was given continuously to 55 patients with a recent history of rheumatic fever during 79 "person-seasons" between 1936 and 1940. Sixty-seven patients with a similar history, who were given no prophylactic treatment, were observed simultaneously as controls during 150 person-seasons. The drug was taken from November through June, usually in

[1] *The Journal of Clinical Investigation*, Volume XVIII, 1939, page 147, abstracted in "The British Encyclopædia of Medical Practice: Surveys and Abstracts", 1939, page 523.

[2] *Annals of Internal Medicine*, February, 1941, page 1416.

[3] *The Lancet*, September 9, 1939, page 581.

[4] "The Prophylactic Use of Sulfanilamide in Patients Susceptible to Rheumatic Fever", *The Journal of the American Medical Association*, February 15, 1941, page 560.

a dose of 1·2 grammes a day. No serious toxic effects were observed. Mild cutaneous eruptions and some drops in the total white cell count, without granulocytopenia, occurred in a few patients. The leucopenia was self-limited in duration and was of no clinical significance. Cultures from pharyngeal swabbings which were positive for β-hæmolytic streptococci were less numerous and showed a lower percentage of organisms among treated patients than among control persons (12·5% of 336 cultures from controls and 4% of 500 cultures from treated patients). While taking sulphanilamide, none of the patients had a major attack of acute rheumatic fever or an acute infection due to the β-hæmolytic streptococcus. One patient who was treated during the winter months, had an acute rheumatic recrudescence in August when he was not taking the drug. Among the control group fifteen major attacks of rheumatic fever occurred and five control persons suffered from other acute illnesses which might have been rheumatic in nature. One person in the control group was admitted to hospital with an acute infection caused by a β-hæmolytic streptococcus. Subacute bacterial endocarditis occurred in two persons in the control group. Four deaths occurred in this group—one from acute rheumatic fever and two from subacute bacterial endocarditis; the cause of the remaining death was uncertain. There were no deaths among those who received prophylactic treatment.

Caroline B. Thomas has now published another paper[1] which must be regarded as of unusual value. She not only adds the observations of eighteen months to those of the four-year period just described, which incidentally she also reviews, but she has correlated her results with those of others working in the same field. In the season of 1940-1941, that is, after the period dealt with by the previous communication, she accepted the prophylactic use of sulphanilamide "as a valuable medical procedure in the management of rheumatic patients" and continued to give the drug to those who wished to continue to take it, and also to any new and suitable patients referred to her. The observation of a control group was abandoned. Of the 25 patients who received the drug in the last year of the four-year study, 21 chose to continue taking it in 1940-1941 and fourteen were still taking it in the winter of 1941-1942. She has therefore had the opportunity of observing nineteen patients who have taken the drug for three years or more. Of these nineteen patients, eleven had taken sulphanilamide for three years, four had taken it for four years, three for five years, and one boy, a member of the original group, was, when Thomas wrote, taking it for the sixth year. This boy first received prophylactic treatment at the age of seventeen years after his fourth attack of rheumatic fever. He is the boy, previously mentioned, who had an acute attack in August after his second season of sulphanilamide while he was not taking the drug. After the August bout of rheumatism the boy insisted on taking the drug summer and winter; for the last four years he has not been without it. When he first came into Thomas's study he had mitral stenosis and aortic insufficiency without decompensation, but the cardiac condition has remained stationary. What is more important is that there is no evidence to suggest that the continuous use of sulphanilamide has had any undesirable effect on his mental or physical state. The other patients who have taken sulphanilamide for long periods of time are stated to be in vigorous physical condition and to be unusually free from minor illnesses. Some of them have taken the drug continuously for two or more years. Thomas's general conclusion, since rheumatic recurrences may come at any time, is that it is wiser to give the drug the year round rather than to give the patient a summer vacation. But the sky of Thomas's achievement has not been entirely unclouded. She reports two cases in which treated patients have had recrudescences of the original illness. The first patient was a girl, aged sixteen years, who started prophylactic treatment after her third attack of acute rheumatism. During her third "season" of taking the drug she married and took a factory job on a night shift. She "found these new adjustments difficult and was fearful and under nervous strain". She had two slight colds with sore throats, but the organisms grown on culture from her pharynx and the erythrocyte sedimentation rate were normal when examinations were made at the usual intervals. Three months after her marriage she was admitted to hospital with chorea and remained there for seven weeks. On no occasion did she have fever and the β-hæmolytic streptococcus could not be recovered from the pharynx. Obviously the chorea was not severe and Thomas remarks that in view of the uncertainty of the ætiological factors contributing to the appearance of chorea, it is possible only to speculate as to whether sulphanilamide prophylaxis will be effective against it. Of the second case much detail need not be given. The patient was a Negro boy of seventeen years who had taken his sulphanilamide fairly conscientiously, missing only an occasional dose. He had a recrudescence with joint manifestations and fever. A few colonies of β-hæmolytic streptococci were grown from his pharynx a few weeks before his illness. The recrudescence was mild and of short duration. This is a genuine failure of the sulphanilamide prophylaxis to give protection.

After giving these details of her own work, Thomas reviews work done by other observers. She refers to Coburn and Moore and then mentions some unpublished work by A. G. Kuttner. Kuttner's work was done in a convalescent home for children recovering from rheumatic fever. She divided the children into two groups, gave sulphanilamide to those in one group and used the others as a control. None of the treated group had a rheumatic recurrence, but 30 of 54 untreated children developed streptococcal pharyngitis, and 14 of these later on suffered from rheumatic recurrences. The importance of this study, as Thomas points out, lies in the fact that both treated and untreated or control groups were exposed to the same environment and had little outside contact—all were equally exposed to β-hæmolytic streptococcal carriers within the convalescent home itself. In the work of all the authors mentioned by Thomas sulphanilamide was the drug used, and during six years several hundreds of children and young adults have received the drug for a total of 648 patient-seasons. Only six have had rheumatic recurrences. Recrudescences among controls have occurred in percentages ranging from 10 to 35. According to this rate the number of recurrences among treated persons should have been 130 rather than six, had sulphanilamide been ineffective. Truly "an impressive body of evidence in favour of this method of treatment" has been produced.

[1] *Bulletin of the New York Academy of Medicine*, August, 1942.

In regard to toxic symptoms it is important to note that when small doses have been used (two groups of authors did this) toxic symptoms have caused less trouble than when larger doses were given. Thomas's dosage has not exceeded 1·3 grammes a day; other authors have given two or even three grammes a day. Only two patients in Thomas's group stopped taking the drug on account of toxic reactions. One boy refused to take it on account of urticaria and nausea; and another had to discontinue it, but was later able to take it again. D. D. Stowell and W. H. Button are recorded as having observed toxic effects in 19 out of 46 patients taking two grammes a day. In 25% of cases the drug was stopped permanently. It has been noted that though the leucocyte count falls sometimes to levels between 2,500 and 4,500 cells per cubic millimetre, a subsequent increase takes place without discontinuance of the drug. The importance of a reduction in the number of leucocytes lies in the fact that it may be an indication of the onset of that rare but grave complication, agranulocytosis. Thomas has collected the records of the available reports of agranulocytosis following sulphanilamide therapy. The cases number twenty-seven and fifteen of them were fatal. None of the cases occurred before the fourteenth or after the forty-ninth day of therapy; most occurred after the eighteenth to twenty-first day. The size of the dose was not a determining factor, nor did the continuous or intermittent taking of the drug have any bearing on the result. Thomas concludes that while injury to the leucocyte-forming mechanism may occur within the first two weeks of treatment, such injury is probably followed by a latent period during which it is impossible to foretell the imminence of agranulocytosis. It is during this period that frequent leucocyte counts should be made. Thomas insists rightly on the rarity of agranulocytosis—she has been able to collect records of only fifteen deaths and sulphanilamide must have been given to hundreds of thousands of patients in the treatment of many different diseases. Most clinicians will agree with her that the risk entailed in giving sulphanilamide seems so much less than the chance of serious rheumatic heart disease developing if treatment is withheld, that the risk must be accepted, and, after proper precautions are taken, disregarded, in order to give the rheumatic patient the best available treatment.

The work described by Thomas and the opinions advanced by her have been set out in some detail, because the journal in which her paper appears is not readily obtainable in this country. It would be safe to describe those carefully controlled observations as comprising one of the most important preventive medicine investigations of recent years. In regard to this statement it must be remembered that it is the recurrence of an acquired infection that is prevented and not the original infection; moreover, the procedure adopted is purely empirical. There is a further point—in view of what is known of the sulphonamide drugs and their actions, the success achieved by the prophylactic administration of sulphanilamide to the subjects of quiescent rheumatic fever is another indication of the probable streptococcal origin of the rheumatic infection. That sulphanilamide is not effective in the acute stage of the disease is not necessarily against the acceptance of this statement. A final conclusion may be drawn, namely, that in the light of all the work done on this subject it would not now be right to form control groups of patients and to withhold from them, for purely experimental reasons, a drug which has been proved capable of producing such striking results as have been reported.

Current Comment.

THE TRANSMISSION OF KALA-AZAR.

Kala-azar is a chronic disease characterized by fever, emaciation, anæmia and splenomegaly. It is caused by infection with *Leishmania*. It occurs in epidemic form in parts of India, China, Russia, Africa and the Mediterranean littoral. In India it affects adults chiefly and is caused by *Leishmania donovani*; while in the countries bordering the Mediterranean it is almost confined to infants and is caused by *Leishmania infantum*. The disease has strange and mysterious features in its endemicity and epidemicity; but at the moment we are concerned only with the transmission of kala-azar in India. For twenty years the sandfly (*Phlebotomus*) has been suspected as the vector. It was shown that the infecting organisms developed with great rapidity in the sandfly's alimentary tract. It was also shown that kala-azar did not occur in places where there were no sandflies. But when it came to the experimental transmission of the infection by sandflies, great difficulties were encountered; indeed for a time all attempts failed. It was found difficult, for example, to keep the delicate insects alive for longer than five days after an infective feed. The disease was transmitted to the Chinese hamster (*Cricetulus griseus*) by injection of an emulsion of crushed infected sandfly, and eventually a hamster was infected by the living sandfly's attack. But attempts to infect man were all futile until last year, when C. S. Swaminath, H. E. Shortt and L. A. P. Anderson succeeded, apparently as a result of a change in the method of feeding the sandflies.[1] Formerly it was the practice to feed the flies on the blood of man or other vertebrates after the infecting feed. In this experiment they were kept alive, according to a technique described by Smith, Halder and Ahmed, by being fed with fruit juice. The flies were kept in tubes in an incubator at a temperature of 28° C. for three or four days; they were then transferred to lamp globes at the same temperature, and were fed with raisins. They were kept in these conditions until a minimum of eight days had elapsed from the time of the infecting feed. Despite these careful measures it was seldom that as many as 50% of the flies survived. Five healthy men who had always lived in and had never left the Khasi hills (a non-endemic area) were selected for the experiment. Every one was infected. Swaminath, Shortt and Anderson pay a tribute to these brave men, each of whom was a volunteer.

Swaminath, Shortt and Anderson suggest two possible reasons for the success of their experiment and the failure of previous ones. The first is the different method of feeding the sandflies. Perhaps feeding on fruit juice enhances the virulence of the organism or increases the heaviness of the infection. The second possible reason is that previously experiments on a large scale were carried out only when the epidemic was on the wane and the organism's virulence was declining. These are matters that will be decided by later research. The great thing is that at last the vector of kala-azar in India has been revealed. We need add no more than the following quotation from an editorial article in the same issue of *The Indian Journal of Medical Research*:

> Twenty years of patient investigation have gone to the forging of this final link in the evidence that *Phlebotomus argentipes* is the insect vector of human kala-azar in India, and the many workers who have taken part in the investigations at different stages and have contributed to the final solution of the problem are to be congratulated on the outcome of their work.[2]

[1] *The Indian Journal of Medical Research*, July, 1942, which has only recently come to hand.
[2] *The Indian Journal of Medical Research*, July, 1924.

Abstracts from Medical Literature.

SURGERY.

Post-Operative Thrombophlebitis and Embolism.

N. W. BARKER AND J. T. PRIESTLEY (*Surgery*, September, 1942) point out that post-operative thrombosis and embolism are not common complications, being found in only 0·96% of surgical operations, but more commonly following laparotomy and most commonly after operations on the female pelvic organs. There appears to be no satisfactory test which will determine which patient is liable to this complication. However, certain predisposing factors can be recognized. These include the presence of carcinoma, diseases of the peripheral veins, severe infections, heart disease, anæmia and blood dyscrasias. The incidence was increased in those patients who had had previous thrombophlebitis or pulmonary embolism (either spontaneously or after a previous operation). This was most marked when the previous incident had been a recent one, within, say, six months. In recent years it has been shown that these complications can be prevented by the adequate administration of heparin. This substance, however, is expensive and somewhat difficult to administer. Recently another substance has been found effective, a preparation known as "Dicoumarin". It is not expensive, and is easily given by mouth. The dosage of the drug must be carefully regulated for each individual by frequent determinations of prothrombin time. Care in this respect will obviate the danger of causing a tendency to hæmorrhage. If such a tendency does arise in the case of the administration of heparin, it may be checked by discontinuing the drug and by the use of protamine. In the case of "Dicoumarin", the effect may be stopped by the administration of fresh citrated blood, which may need to be repeated. The authors suggest that in certain patients in whom the present condition or the previous history, as indicated above, would suggest the likelihood of thrombosis or embolism occurring after operation, prophylaxis should be considered by the administration of heparin or "Dicoumarin".

Wounds of the Vascular System.

WRITING from the Surgical Vascular Clinic, New York Post-Graduate Hospital and Medical School, G. H. Pratt (*The American Journal of Surgery*, July, 1942) deplores the neglect of teaching in regard to the management of vascular problems in surgery. The emergency control of arterial bleeding is possible by digital pressure, and this has been maintained at times for several days. The application of a clamp to a large vessel is more satisfactory than the application of a tourniquet. When the use of a tourniquet is necessary, it should be applied as close as possible to the wound. When the use of a tourniquet has been necessary, operation should be performed at the nearest hospital. Amputation may be required following vascular injuries, but it should be performed only as a last resort. When an artery has been severed, an end-to-end suture can often be accomplished. Suture material should be fine and the needles minute. The needle should not enter the intima. Very few sutures are necessary. A muscle or fascial graft may be used to reinforce the suture line. Flexion of the limb may allow a defect in length of the artery to be overcome. A venous transplant has been successful in many cases. Arterial thrombi, present in 50% of injuries to arteries, should be removed prior to suturing. When repair is impossible and ligation of the vessel is unavoidable, loss of the limb does not necessarily follow. The femoral artery may be ligated in 18% without subsequent gangrene, and this may be decreased to approximately 8% by ligation of the accompanying vein. After a severe vascular injury the physiological action of the collateral vessels is to go into spasm. Therefore, after the major vessel has been repaired or ligated, efforts should be made to dilate these collaterals. In this, external heat may be helpful, and is best maintained between 88° and 96° F. Repeated elevation, levelling and lowering of the limb provide a mild vascular massage. The author insists that the patient should not be allowed to smoke, and he protests against the common practice of immediately giving a wounded soldier a cigarette. Nicotine causes spasm of small vessels, and at this stage may determine inadequacy of the already embarrassed collateral circulation. Certain smooth muscle dilators may be of value, and one of the most useful is alcohol in the form of whisky. In the case of the lower limb, blocking of the sympathetic nerves is often helpful, and this may be done by a local anæsthetic injection into the perivertebral region, the needle being entered in the lumbar region four centimetres lateral to the mid-line and between the transverse processes. Diagrams show the details of the technique.

Thrombocytopenic Purpura.

J. R. PHILLIPS AND M. A. ZIONTE (*The American Journal of Surgery*, July, 1942) discuss the classification, diagnosis and treatment of thrombocytopenic purpura. It is of interest to the surgeon because, in the idiopathic cases, splenectomy is the best method of treatment. Thrombocytopenia occurs as a symptom in many diseases, for instance, acute and chronic infections, drug intoxications, advanced malignant disease with metastases to the bone marrow, and in blood dyscrasias such as leuchæmia, aplastic anæmia, pernicious anæmia and others. It is most necessary, before operation is considered, that the condition be ascertained to be one of idiopathic thrombocytopenia, and not one of the symptomatic variety. In the treatment of the idiopathic variety, many types of medical management have been tried, but for the most part with success only in the hands of their originators. Many patients, especially children, have spontaneous remissions, and may then remain well for the rest of their lives. In acute cases when the symptoms fail to abate spontaneously the surgeon may be forced to operate in order to attempt to save life. But the operation is hazardous in these cases, and the mortality is from 70% to 80%. In the chronic cases, the results are excellent, and the mortality is only 8%. During operation, it is very necessary to be certain that no accessory spleen is left behind, as recurrences are seen when the accessory organ hypertrophies.

Bilateral Bronchiectasis.

BRIAN BLADES AND EVARTS A. GRAHAM (*Surgery, Gynecology and Obstetrics*, October, 1942) discuss the surgical treatment of bilateral bronchiectasis. In at least 30% of patients with bronchiectasis the disease is bilateral. Experience with 16 cases suggests that in young subjects surgical treatment should be recommended if the two upper lobes are free of disease (the lingula of the left upper lobe being regarded as the left middle lobe). When the contralateral disease is slight, unilateral lobectomy may give considerable relief. The upright position at operation is employed to lessen the danger of suffocation, and to allow unhurried closure of the bronchus.

Management of Cerebral Trauma.

ELLIOTT C. CUTLER AND ROBERT D. WHITFIELD (*The American Journal of Surgery*, July, 1942) report their experiences with 396 cases of cerebral trauma seen over a period of seventeen years. The main consideration in these cases is the injury to the underlying brain rather than the bony injury. The problem of treatment is largely the problem of the control of intracranial pressure. An injured brain reacts in the same way as other tissues, but the effect is modified by the fact that the brain is enclosed in a closed box. If swelling of cerebral tissue occurs, the increased space necessary to accommodate it must be provided by a diminution in the space occupied by blood in the skull cavity. As the swelling increases, there is therefore a progressive anoxæmia of the cerebrum. The intravenous injection of hypertonic solutions will withdraw fluid from the œdematous tissues and thus reduce the intracranial tension. At first hypertonic saline solution was used, but numerous petechial hæmorrhages were found in the cerebral tissue following its use. Later, hypertonic glucose solution was used, and still later, hypertonic sucrose solution was found even more suitable. Another method of value is dehydration produced either by restriction of fluid intake or by watery catharsis. Yet another method is the withdrawal of cerebro-spinal fluid by lumbar puncture, but generally speaking this method has been condemned. In the presence of gross damage it may lead to further medullary embarrassment and death. But the authors use the method widely as a diagnostic procedure. Spinal fluid pressure is measured, and a small amount of fluid is withdrawn for examination. The paramount consideration in the treatment of these patients is complete rest in bed until one full week after all symptoms have disappeared. Moderate limitation of fluids is recommended, and occasional magnesium sulphate catharsis. X-ray films are taken, but unless there is some suspicion of depression, this may often be delayed for some days. Operation is resorted to in the presence of certain indications, namely, (*a*) increasing coma after a period of several hours' observation; (*b*) the appearance of focal neurological signs indicating a rapidly

growing extradural or acute subdural hæmatoma; (c) non-improvement of the patient's condition after forty-eight hours of conservative treatment; (d) compound fractures of the skull. The percentage of patients coming to operation has decreased considerably over recent years, and this has been accompanied by a corresponding fall in the gross mortality. The authors insist that in the treatment of injuries of this type, accurate history taking, painstaking examination of the patient and conscientious observation of his course are absolutely essential.

The Healing of Cutaneous Wounds.

B. E. BRUSH AND C. R. LAM (*Surgery*, September, 1942) report the results of some experimental work on wound healing. Much has been written on the subject of the influence on the rate of healing of wounds of various substances, either applied locally or taken by mouth. There appears to be definite evidence that certain substances taken internally may have such an influence, for instance, vitamin *C* appears to play such a part. But evidence that substances applied locally can have any influence is much less convincing. The authors review shortly some of the claims which have been advanced with regard to a great variety of such substances. For the most part, these claims are not well substantiated. At the suggestion of Dr. Alexis Carrel and Dr. Roy D. McClure, the authors therefore decided to carry out a series of experiments in order to test as scientifically as possible the influence on wound healing of certain substances, some of which are in common use as wound dressings. These include various strengths of chloramine in the form of a paste, "Scarlet red" ointment, Peruvian balsam, urea crystals, glycerin, 2% pectin solution, commercial vitamins *A* and *D* ointment, and chlorophyll ointment. The wounds used were made on the prepared skin of the abdominal walls of guinea-pigs. Two wounds were made on each abdomen, one being used as a control and one as the test wound. Twelve guinea-pigs were used for each substance tested. Dressings were changed every two days, and at each dressing the wound was measured by making a tracing. The results are recorded. They show that except in the case of the higher strengths of chloramine paste and in the case of the urea dressings, there was no appreciable effect on the rate of healing. With the substances mentioned, however, there was delay. This was most marked with the urea, where the average time for healing of the test wounds was 20·6 days, while the average for the control wounds on the same animals was only 12·3 days. It should be pointed out that these experiments concern healing of primarily non-infected wounds in apparently healthy animals.

Pulmonary Abscess.

RICHMOND L. MOORE (*Annals of Surgery*, September, 1942) discusses pulmonary abscess as a surgical problem, and reports the results of 78 cases in which surgical treatment was adopted. According to the pathological change the abscesses are classified as complicated and uncomplicated; and according to odour as fœtid and non-fœtid. Complications include multiple abscesses, empyema, fibrosis and bronchiectasis. The terms "acute" and "chronic" are not used. The majority of fœtid abscesses are due to aspiration, whereas most non-fœtid abscesses are of embolic or post-pneumonic origin. In the former anaerobic organisms predominate, in the latter aerobes. Free drainage is the most important single factor in treatment. This may occur spontaneously through a bronchus or be established surgically. Diagnostic aspiration may infect the pleural cavity and should be avoided. Pulmonary abscess, especially of the fœtid type, is a surgical disease. If conservative treatment fails, early free drainage is essential to avoid development of complications and the higher operative mortality and less satisfactory late results in the complicated cases.

Technique of Appendicectomy.

HUGH AUCHINCLOSS (*Annals of Surgery*, September, 1942) attributes the lower death rate from appendicitis in the large hospital to better technique and experience. Pre-operative recognition of the position of the appendix enables the incision to be more accurately placed. The McBurney incision, or in cases of doubt a mid-line incision, is recommended. The right rectus incision is condemned. The incision in the skin is often unnecessarily long. With adequate retraction the surgeon should "see" before "feeling". The appendix stump should be inverted without ligation. The badly infected appendix should always be removed unless there is convincing evidence to the contrary. When the surgeon is in doubt he should use drainage, but in the presence of serious infection tight closure round the tube is dangerous. In these cases the wound should be kept partly open by the use of light packing. The local use of sulphanilamide or sulphathiazole is advocated.

The Bleeding Peptic Ulcer.

M. JORDAN THORSTAD (*Surgery*, December, 1942) reviews the literature on the subject of treatment of bleeding peptic ulcer. The incidence of this complication in peptic ulcer seems to be about 18% to 20%. It is four times as common in men as in women, and reaches its peak in the fifth decade of life. An increase in the incidence of bleeding peptic ulcer has been reported in England since the outbreak of war. Nervous and emotional factors appear to play an important part in provoking this complication. Patients rarely die during the initial massive hæmorrhage. Blalock found experimentally that death is more often due to shock than to exsanguination. Exhaustion, accentuated by severe restriction of diet, may be a factor in mortality. Initial treatment must be for the shock. Rest, quiet and warmth are essential, and may be supplemented by adrenal cortex extract, morphine given sparingly, and oxygen. Transfusions are administered with care. The starvation diet has largely been replaced by the more liberal feeding suggested by Meulengracht. The introduction of this dietary *régime* has materially reduced the mortality from bleeding ulcer. During the past ten years, there has been a trend towards operative treatment in some of these cases. Finsterer first used early operative treatment, and among patients on whom he operated within forty-eight hours of the onset of bleeding, he claimed a mortality of 5·1%; but when operation was performed later than this, the mortality rose to 29·7%. Many surgeons have attempted to define the conditions under which operation should be performed, but there does not appear to be any generally accepted standard. Many series of results of the mortality of surgical treatment have been published, varying from Finsterer's figure of 5·1% in early cases, up to 100% in others. No series approaches the low mortality rate which Meulengracht claims for his dietary treatment, namely, 1% to 2%. The author reports a series of 284 patients admitted to the Harper Hospital during the years 1931 to 1941. Of these, fifteen were operated on, seven of them having massive hæmorrhage; of these one died. Among 138 patients with acute massive hæmorrhage treated by starvation *régime*, the mortality was 13·7%. Among 70 patients with acute massive hæmorrhage treated by prompt and frequent feeding, the mortality was 2·8%.

The Delayed Classification of Burns.

EDWIN P. LEHMAN ("Editorial", *Surgery*, October, 1942) indicates the impossibility of judging the depth of a burn in relation to full or partial thickness of the skin when first seen. The measurement of the area affords prognostic data as to survival, while the estimation of depth or "degree" affords data as to the time of healing and the necessity for skin grafting. The author suggests a simple practical classification of burns into three groups. The first group comprises the most superficial burns in which there is no important loss of substance. Diagnosis is immediate. Prognosis and treatment present no great problems. In the second group there is loss of substance involving part of the thickness of the skin. Diagnosis is impossible until sloughing occurs, healing is slower, but grafting is unnecessary. In the third group the loss involves the whole thickness of the skin. Diagnosis is impossible until sloughing occurs, and skin grafting is necessary.

Sliding Hernia.

CLAUDE C. BURTON AND CARL BLOTNER (*Annals of Surgery*, September, 1942) comment upon the scantiness of the literature dealing with sliding hernia. In sliding hernia part of the wall is formed by a viscus which in its normal position is only in part covered by peritoneum. Sliding herniæ may be classified as intrasaccular and parasaccular or alternatively as right colon, left colon and bladder. The mechanism and diagnosis are discussed. In treatment three methods of repairing the sac are illustrated and complications that may occur at this stage are described. The methods of repairing the wall are described as triplicate fascial closure, and as requiring the use of fascial sutures, Cooper's ligament and pedicled fascial graft. The first two were the most commonly employed in a series of 43 primary sliding herniæ with a total recurrence rate of 6·9%. By triplicate fascial closure is meant suture of transversalis fascia to Poupart's ligament, and imbrication of external oblique with extra-aponeurotic displacement of the cord.

Naval, Military and Air Force.

CLINICAL MEETING AT AN AUSTRALIAN GENERAL HOSPITAL.

A CLINICAL meeting was held on September 26, 1942, at the 121st Australian General Hospital. In addition to the members of the medical staff a number of visitors were present, including Colonel F. H. Beare, D.D.M.S. Norforce, Colonel N. D. Barton, A.D.M.S. Field Force, and representatives of the Royal Australian Navy, the Royal Australian Air Force and the United States Army Medical Service.

Splenomegaly.

CAPTAIN H. B. KAY showed a number of patients to illustrate some of the causes of splenomegaly encountered in the Northern Territory.

Benign Tertian Malaria.

Captain Kay's first patient was a man, aged twenty-six years, who had been admitted to the hospital on August 28, 1942, with a history of fever and chills associated with headache and backache of one day's duration. He had had a similar attack three weeks previously whilst on the Roper River; this had subsided under treatment with quinine. On examination, the patient's temperature was 98·8° F.; the spleen was firm and easily palpable. On August 30 examination of a blood film disclosed *Plasmodium vivax*. A full course of treatment was instituted. Examination of a further blood film on September 20 revealed no parasites; but the spleen was still easily palpable.

Secondary Syphilis.

The second patient shown by Captain Kay was a man, aged nineteen years, who had been admitted to hospital on August 18, 1942, with a history of a penile sore of two weeks' duration. The appearance of the lesion was suggestive of primary chancre; but dark-ground examination failed to reveal the presence of *Spirochæta pallida*. Examination of material aspirated from a left inguinal lymph gland gave negative results, and the Kline test failed to produce a reaction. On September 13 the patient complained of headache and fever. On examination, he was found to have a temperature of 100° F.; a fine, macular rash was present over the trunk and the spleen was palpable. Numerous small shotty lymph glands were palpable. On September 18 the Kline test produced a reaction.

Infective Hepatitis.

Captain Kay's next patient was a man, aged twenty-eight years, who had been admitted to hospital on August 31, 1942, with a history of shivering and fever, headache and anorexia, of two days' duration. The patient had had no previous illness and had been in the area for only three weeks. On examination, he was found to have a temperature of 101° F.; his spleen was palpable and firm, but his liver was not palpable. No icterus was present. On September 4 an icteric tinge of the skin and conjunctivæ was noticed; this cleared up in ten days, but the spleen remained palpable. A blood examination revealed that the leucocytes numbered 9,800 per cubic millimetre, 55% being neutrophile cells, 41·5% lymphocytes, 2·5% monocytes and 1% eosinophile cells.

Another patient, aged twenty-nine years, had been admitted to hospital suffering from anorexia, headache and backache. On examination, mild icterus was present; the spleen was palpable and firm and the liver edge was palpable and tender. The jaundice cleared up in two weeks and the liver enlargement subsided; but the spleen was still palpable. A blood examination revealed that the leucocytes numbered 2,500 per cubic millimetre, 43% being neutrophile cells, 50% lymphocytes, 5% monocytes and 2% eosinophile cells. On September 24 the leucocytes numbered 8,200 per cubic millimetre.

Bacillus Flexner Dysentery.

Captain Kay's fifth patient was aged twenty-three years; he had been admitted to hospital on September 10, 1942, with the diagnosis of dengue fever, suffering from headache, backache and fever. On examination, he was found to have a temperature of 103° F., and a fine macular rash was present over his body; the axillary glands were palpable, but neither the spleen nor the liver was palpable. Examination of the blood revealed no parasites. On September 20 the patient suffered from diarrhœa with the passage of blood and mucus; his temperature was 102° F., and the spleen was palpable and firm, but not tender. Culture of the fæces resulted in a growth of *Bacillus dysenteriæ* Flexner. On September 22, examination of the blood disclosed that the leucocytes numbered 8,400 per cubic millimetre, 48·5% being neutrophile cells, 50% lymphocytes, 1% monocytes and 0·5% eosinophile cells. By September 24 the diarrhœa had subsided and the spleen was not palpable.

The sixth patient shown by Captain Kay was aged twenty-three years. He had been admitted to hospital on September 5, 1942, with a history of chronic cough of two months' duration, for which an investigation had been carried out at another hospital. Radiological examination of the chest revealed no abnormality, and examination of the sputum gave negative results. On September 12 the patient suffered from diarrhœa, with the passage of fluid stools containing neither blood nor mucus. His temperature was 102° F.; the spleen was palpable, but the liver was not. Examination of three blood films failed to reveal any parasites. The leucocytes numbered 11,100 per cubic millimetre of blood, 57% being neutrophile cells, 29% lymphocytes, 10·5% monocytes and 3·5% eosinophile cells. The hæmoglobin value was 14 grammes per 100 cubic centimetres, and the erythrocytes were of normal appearance. On September 20 the spleen was still palpable; the liver was not palpable, but there was an area of tenderness under the right costal margin. No icterus was present, and there were no enlarged lymph glands. The Weil-Felix test failed to produce a reaction. Captain Kay said that the lesion was undoubtedly infective hepatitis with slow onset of icterus.

Finally, Captain Kay showed a patient, aged twenty-three years, who had served in the area for ten months. He had been admitted to hospital on September 4, 1942, for investigation of headache. Six months earlier he had been treated in hospital for what was believed to be an attack of dengue fever; but no records were available at the time of the meeting. Since that attack the patient had had dragging pain under the left costal margin. He had had no previous illnesses, and had never been out of Australia. On examination, the spleen was easily palpable, firm and not tender; the liver was not palpable. No enlarged lymph glands were found. Examination of the blood revealed that the erythrocytes were of normal appearance; the hæmoglobin value was 14·5 grammes per 100 cubic centimetres, and the fragility of the erythrocytes was normal; the leucocytes numbered 8,000 per cubic millimetre, 61% being neutrophile cells, 26·5% lymphocytes, 5·5% eosinophile cells, 0·5% basophile cells and 6·5% monocytes. Radiological examination of the chest revealed no abnormality, and no abnormality was detected in the urine. The Kline test failed to produce a reaction. No malarial parasites were detected in repeated examinations of blood films.

The Transport of Wounded in Plaster of Paris Splints.

CAPTAIN R. S. LAWSON showed a series of patients with wounds and fractures which had been treated by immobilization in plaster of Paris. He then discussed the transport of wounded in plaster of Paris splints.

Captain Lawson said that the general advantages of the treatment of compound fractures in closed plaster splints were so well known that it was unnecessary to dwell on them; but they could be summarized briefly in the following terms: (i) absolute immobilization with all its attendant benefits was assured; (ii) exogenous infecton and particularly cross-infection was excluded; (iii) the method was simple—in effect, one splint served for any part of the body; (iv) repeated dressings, with all the labour, expense, pain and hazards which these might entail, were eliminated.

Captain Lawson went on to say that the first matter to be settled was the stage at which the method might be usefully applied. Plainly, it was not in the regimental aid post or the advanced dressing station nor in a main dressing station in its normal way of functioning; but the main dressing station had been made responsible for operations on battle casualties in special circumstances, and, of course, that was the crux of the matter—namely, that plaster of Paris would be applied after the operation had been performed, wherever that might be; it was most often carried out in a casualty clearing station or otherwise by a surgical team attached to a field ambulance. Plaster of Paris was applied after excision of the wound. It was used chiefly for compound fractures, but it could be most valuable in many cases of soft tissue wounds not associated with bony damage. In either case, its primary function in these circumstances was undoubtedly that of a splint. Obviously,

good surgical treatment before the plaster was put on was essential, and the most elegant plaster of Paris splint would not make up for deficiencies in that respect. In most cases after operation sulphanilamide and a "Vaseline" dressing would be applied.

Captain Lawson then said that in the application of a plaster of Paris splint the first rule was to play safe. It should always be borne in mind that the patient might have to be sent off the following day down a long line of evacuation. In fact, those were just the conditions under which the method was of particular value, and which had made it so popular. As a corollary, it followed that the patient might have to travel perhaps for some days without attention to the plaster splint, either because there was no one at hand to judge whether things were going wrong, or because there was no opportunity to correct matters if they were. Risk of constriction had to be avoided above all, and it should be remembered that plaster splints were never foolproof. One soldier in the Western Desert had suffered a simple fracture of the tibia. After its reduction, a non-padded plaster splint was applied. Gangrene of the foot supervened, necessitating amputation below the knee. Captain Lawson said that he was not decrying the use of non-padded plaster splints in ordinary circumstances; but in the primary treatment of war wounds they should be used with great circumspection, if at all. An accurate reduction of the fracture was not essential. Naturally, every effort should be made to obtain the best possible position, but some degree of angulation or rotation or even some overlap were unimportant in the circumstances and perhaps unavoidable. Correction of those minor malalignments was a job for the radiologist and the surgeons at a base hospital. For example, most plaster spicas were removed when the casualties reached their ultimate destination at the base hospital from the Western Desert, and the limbs were put up in Thomas splint extension. That was what had been anticipated at both ends, and it brought out the point that the primary function of the plaster splint was to act as a high-class temporary splint. At a certain base hospital working in a forward area and handling battle casualties direct from the front, the standard of fracture reduction was, of course, very high; but Captain Lawson was referring to the more usual circumstances in which the casualties were treated in a casualty clearing station without the same facilities for treatment or capacity for keeping them. At one such casualty clearing station to which he had been attached, the X-ray plant was bombed early, and hundreds of casualties were subsequently treated without the aid of radiology and evacuated usually on the day following operation.

Captain Lawson went on to discuss individual plaster casts. He said that with regard to the lower limb, wounds of the foot, ankle and lower third of the leg required a right-angled plaster cast up to the knee. If the wound or fracture was above the lower third of the leg, the plaster must be extended to the mid-thigh. Wounds of the knee-joint and above required a plaster spica. The spica should extend from the toes to above the iliac crests, which should be gripped on both sides. The ideal spica from an orthopædic point of view extended partly down the opposite thigh. This was unnecessary in primary war surgery. The hip spica was not so formidable as it sounded, and with a practised team it should not take longer than fifteen minutes to apply, though it certainly made inroads on the stocks of plaster. An orthopædic table was a luxury which could not be expected; but some sort of pelvic rest was an important adjunct. Failing this, a tent pole lashed to the end of the operating table could be made to serve, and when extension was not required an upturned basin under the patient's buttocks would suffice; but three assistants (orderlies) were needed. There had been some controversy concerning the respective merits of the Thomas splint and the plaster spica in these cases, particularly for fractured femur. Both methods were valuable, and Colonel W. A. Hailes in a recent article had given his approval to both methods, though he gave first place to the Thomas splint. Captain Lawson said that in his opinion, when the line of evacuation was relatively short and well-defined, the Thomas splint was to be preferred, and he understood that that was the experience in the Syrian campaign. When the line of evacuation was long and uncertain the plaster spica was probably the method of choice. For example, some casualties evacuated from the Western Desert had a sea voyage followed by a long train journey and perhaps three or four stages after that, including numerous short "carries" in motor ambulances before they reached their final destination as long as ten or twelve days later. He believed that, if the plaster spica did a good job for that distance, the army need not begrudge a few dozen plaster bandages, even if the plaster was removed on the casualties' final arrival, as it frequently was.

Captain Lawson said that one positive contraindication to the plaster spica was a threat of hæmorrhage beneath it. He instanced the case of a wounded soldier, who was seen by him in transit. The soldier had been treated in a plaster spica for a compound fracture of the left leg and a large flesh wound of the left thigh. On his arrival, blood was oozing from the top of the plaster spica and his pulse rate was rising. The spica was removed down to the knee, and a large wound was revealed with a short length of the intact femoral artery exposed in Hunter's canal. After receiving a blood transfusion the patient was evacuated in a Thomas splint, with a tourniquet ready in place around the upper part of the thigh.

Referring to the upper limb, Captain Lawson said that wounds of the hand and wrist required a plaster splint up to the elbow. Wounds above the lower third of the forearm required a right-angled plaster splint including the arm. For wounds of the arm the plaster splint could be extended in the form of a cap over the shoulder, and in addition a gauze or plaster of Paris bandage could encircle the limb and trunk. Shoulder wounds could be similarly treated, or they could be put into a shoulder spica. Captain Lawson said that there had been some controversy about this plaster spica, and the following objections had been raised to its use. (i) It was held that the casualties wearing this spica could not be fitted into a motor ambulance. It was true that the patient must occupy the upper berth with the injured side towards the centre; but that was a matter which could be easily arranged. (ii) It was stated that the shoulder spica could not be applied under general anæsthesia. That was untrue, for he had used it in civil practice for such conditions as osteomyelitis of the upper end of the humerus and had applied it with the patient still under ether anæsthesia. (iii) It was held to be unnecessary. With that view Captain Lawson largely agreed, but there was an occasional case in which it had a value. Two such cases had occurred in his experience. One was that of an Australian soldier with machine-gun bullets through the head of his humerus and the scapula, and the other was that of an Italian prisoner of war with his posterior axillary fold blasted away by high explosive.

Finally, Captain Lawson said that there were certain cases of amputation in which plaster of Paris could be used with benefit. This was justifiable only when one was absolutely confident of the hæmostasis. It was well known that those who had undergone recent amputations travelled badly, and the support of a plaster of Paris splint in suitable cases would minimize the shock and pain of travelling. In conclusion, one could say that this method of transport of the wounded was one of the major advances that had been made in the handling of battle casualties in the present war.

Backache from Various Causes.

MAJOR N. J. BONNIN showed a series of patients suffering from backache due to the following causes: arthritis of the Marie-Strümpell type, fibrositis, sacro-iliac strain, lumbo-sacral strain associated with a postural lordosis and an incompletely developed fifth lumbar vertical arch, sciatica due probably to ruptured intervertebral disks, and osteo-arthritis of the hip. Major Bonnin then discussed the subject of low backache. He said that in the time available, it was not possible to deal in any detail with the subject of backache, or for that matter in detail with any single aspect of the subject, and he would simply make a few remarks and suggestions which might be of use to those regimental medical officers and others who had to deal with the large number of soldiers who "reported sick" with that common complaint. The causes of backache could be classified into extrinsic and intrinsic. The extrinsic causes included disease of almost any thoracic, abdominal or pelvic viscus. Such disease was discovered or eliminated by ordinary general examination. The intrinsic causes, and by those Major Bonnin meant diseases affecting the structures of the back itself, could be classified into four groups: (i) specific infections (pyococcal or tuberculous), (ii) neoplasms (primary or secondary), (iii) trauma, (iv) fibrositis or arthritic disease. In regard to the first two, it had to be remembered that those present were dealing with a selected group of males in the twenty to forty years age group—they were above the common age for Pott's disease and below the age for malignant disease—and it was safe to say that in the army those were extremely rare conditions, and usually not even thought of in the differential diagnosis. However, continuous pain (not necessarily severe), constant location of the pain, the same local rigidity on repeated

examination and progression of symptoms, all called for an X-ray examination.

With regard to trauma, Major Bonnin said that he did not propose to speak about fractures, except to remind those present that constant, localized pain, rigidity and deformity, perhaps following severe trauma, might be due to the missed fracture of Kümmell's disease. The type of trauma with which he was concerned was the milder trauma, which was often due to active movement, and which damaged ligament or tendon, and the recurrent or chronic trauma due to poor posture. It was with those cases, and those of the fibrositic and arthritic group, that he proposed to deal.

Major Bonnin went on to say that in the majority of cases it was the lower lumbar region that was affected. That region was one of considerable mobility and took the whole weight of the trunk. In that area there were a series of intervertebral joints, each comprising a main anterior joint between vertebral bodies and a pair of lateral joints between articular processes, and the sacro-iliac joints were in that region. About those joints there were many ligaments, and over the ligaments were a multitude of muscles and tendons—all structures exposed to trauma and liable to fibrositic disease. The lumbo-sacral joints were the most vulnerable, for not only was that the region of maximum strain, but they were more mobile than the other lumbar joints, and congenital anomalies were common.

Referring to the ætiology and pathology of fibrositis, Major Bonnin said that they were not understood; but fibrositis was a localized low-grade inflammatory process which might go on to subsequent fibrosis. Trauma to a tendon or ligament commonly resulted in a tearing of some fibres. Consequent pain and muscle spasm caused an abnormal posture or limitation of movement. That led to malunion of the torn fibres by scar tissue which might involve nerves or nerve endings. Later, a normal movement would cause a drag on that scar, with consequent pain. An abnormal posture—for example, lordosis—threw a continual strain on certain ligaments and muscles and rendered them liable to repeated mild trauma. Trauma to cartilage, usually a compression beyond its limit of elasticity, resulted in damage probably of the nature of a crack or fracture. That was followed rapidly by a degenerative process, with fibrillation and softening of the damaged area. Regeneration with rest was slow, and in the absence of rest, the cartilage would be fragmented, or absorbed, or both, and replaced by fibrous tissue. Major Bonnin went on to say that in a great many of these cases of fibrositis, arthritis, or chronic trauma, a septic focus was present somewhere, and it in some way acted as an ætiological factor; elimination of the focus was necessary if a cure was to be obtained. The common sites of sepsis were the teeth and tonsils and occasionally the prostate. The large bowel was also blamed; but he doubted if many backaches were relieved by "number nines".

Major Bonnin further said that beside anatomical and pathological considerations there was another factor that had to be taken into account in almost every case, and that was the psychological element. Even in civilian life, and when there was a strong incentive to return to work, the patient was inclined to exaggerate, as was usual when a complaint was subjective rather than objective. In the army the incentive was apt to be all in the other direction; and especially was that true in the Northern Territory, where so many of the men affected had one eye fixed on the south and the other swivelling about looking for a means of transport. That psychological factor had to be continually allowed for and dealt with, both in the making of the diagnosis and in the carrying out of the treatment. It seemed to Major Bonnin a cardinal principle that those patients should be dealt with expeditiously. Indecision, delay, or "passing the buck" might start a neurosis or perpetuate an incipient one. If the medical officer showed that he was puzzled or uncertain in his dealing with such men, they were likely to think that they had some serious condition (or occasionally a "good line" for "lead swinging"), and their confidence was permanently lost.

Major Bonnin went on to say that when a case of backache was being dealt with, it had first to be accepted that no case could be properly investigated in less than ten to fifteen minutes. It was no use trying to deal with these patients on a "sick parade"; the patient should be made to wait or to report back when time was available. The history was most important and often took a long time to elicit. Particular attention should be paid to three points: (i) the exact mode of onset of the first attack and of subsequent attacks, (ii) the movements or circumstances which brought on the pain, (iii) the exact location of the pain. Major Bonnin said that he could not too strongly stress the point that an examination of the back was almost worthless unless the patient was stripped. Shirt, trousers, puttees, boots and socks had to come off and the patient to be stark naked. The first thing to do was to look at the patient, and that might supply more than half the answer. The inspection was by far the most important part of the examination.[1] It had to be detailed and prolonged, and it could not be carried out unless the patient was naked. The posture, the contours of the spine in detail, and the general musculature should be noted. The patient should then be made to carry out movements in the standing position; he should be made to touch his toes, hollow his back, bend left, bend right, twist left and twist right, and at the end of each movement the examining medical officer should take the patient's shoulders and push him a little beyond his extreme range of movement. His range of movement, and any restriction of movement and in what part of the spine it occurred should be noted; the movement that caused pain should be noted exactly. The patient should then be made to lie on his back on a table or a couch and the hip joints put through their full range of movement—flexion, abduction, adduction, internal and external rotation. In testing flexion the knee was, of course, fully flexed. The sacro-iliac joints should next be wrenched. The patient should be drawn towards the examiner so that his spine was almost on the edge of the table and his shoulder and buttock projected over. The hip and knee of the leg farther from the examiner were flexed so that the patient's thigh was pressed on his abdomen and his knee was near his chin. Next, the thigh, which was hanging over the edge of the table, was fully extended, and the examiner placed one hand on each knee and pressed down forcibly. This manœuvre put the lumbar part of the spine in a neutral position of partial flexion and fixed the pelvis. The other thigh was then hyperextended, so that the ileo-femoral ligament of Bigelow was made tense and the femur and ileum became locked as one bone. The final downward pressure on both knees then rotated the iliac bone on the sacrum at the sacro-iliac joint. A definite, though small, normal movement took place at this joint—a movement of rotation centring about the second piece of the sacrum. The manœuvre was then repeated on the opposite side. If the trouble was located in the sacro-iliac joint, the patient would complain of acute pain in the back over the joint on the side of the extended thigh, and the medical officer could be reasonably certain that if the patient did not complain of such pain the sacro-iliac joints were not at fault. If there was a complaint of some pain in the back on the side of the flexed thigh, the trouble was probably in the lumbo-sacral region, and this would be confirmed later.

Major Bonnin pointed out that thus far the hip and sacro-iliac joints had been eliminated as the cause of the pain. The lumbo-sacral and interlumbar joints were more difficult to examine, because it was impossible to wrench one joint at a time, and a deduction had to be made from a series of observations upon the painful movement, the location of the pain and the tender points. Major Bonnin then described the next procedure to be carried out. The patient was still on his back, and his sacro-iliac joints had just been wrenched. The medical examiner should then take hold of both calves, and with knees half flexed swing both legs up until the patient's knees were near his ears. This manœuvre fully flexed the lumbar part of the spine. The patient's legs should then be lowered, and with hands on the ankle the examiner should flex the right leg as far as possible with knee extended; this manœuvre stretched the hamstrings. The examiner, retaining his hold on the ankle, should then with the other hand hold the patient's right shoulder against the table, and allowing the knee to flex, force the patient's leg across his body until the knee was nearly touching his left shoulder. This manœuvre flexed the lumbar part of the spine and threw a twisting force on it, especially on the lumbo-sacral region. The movements should be repeated with the left leg. Before the patient was turned over, his knee jerks should be tested. The patient should be instructed to turn onto his face, and the examiner should watch how he did it; the "lead-swinger" might give himself away. The whole region of the back and buttocks should be palpated and a search for tender points made with deep pressure. Major Bonnin went on to say that the medical examiner should then pass his forearm beneath the patient's thighs just above his knees and raise his thighs, his pelvis and the lower part of his abdomen completely off the table. The examiner should place his other hand over the lower lumbar part of the patient's spine and press firmly down, thus forcibly extending the lumbar part of the spine; he should then lower the patient's legs and hyperextend first one and then the other thigh, pressing down firmly each time over the

[1] The following examination was demonstrated as it was described.

upper part of the sacral region. The strain occasioned by this movement fell first on the hip joint and then on the sacro-iliac joint; but these had been previously examined. Above these joints there was a force of extension and rotation on the lumbo-sacral and interlumbar joints; the strain fell especially on the lumbo-sacral joints or on a transverse lumbo-sacral joint if it was present. Finally, the medical examiner should test the patient's ankle jerks, and if there was radiating pain in the legs, he should test his sensation to light touch. The examination should be completed by an inspection of the teeth and tonsils, and if there was any history or suspicion of old gonorrhœa, or if there was pain of the sciatic type, a rectal examination should be made. Finally, out of the corner of his eye the medical examiner should watch the patient put on his boots; a remarkable flexibility of the spine was sometimes demonstrated.

Major Bonnin then said that the examination he had described was suggested as a suitable routine examination of a patient complaining of backache. Although it took a long time to describe, in practice it did not take long to carry out once the routine had been learnt, and it would give most of the information obtainable clinically. There were, of course, many other useful manœuvres and many tests and tricks for use in special cases; but they were mainly of a confirmatory nature. The interpretation of the signs elicited was not always so easy. While the examination was in progress, it was essential to carry a mental picture of the anatomy of the region and to visualize what was happening under the patient's skin. When pain was elicited the patient should be asked to demonstrate a pin-point localization. Such pain was in general located accurately over the diseased structure. When the examination was complete, an accurate diagnosis would have been made in quite a large number of cases, and it would be possible for the medical officer to hazard a fairly shrewd guess in most of the others. He would be in a position to decide whether the patient was to be retained or evacuated, and if he was to be retained, what treatment to apply. What was most important, the medical officer was in a position to talk to the patient, and had taken the first essential step in dealing with a potential neurosis. Major Bonnin said that he was well aware of the orthopædic axiom that no examination of the back was complete without an X-ray examination. That was all very well for the orthopædic surgeon in his chromium-plated clinic, but not much use to regimental medical officers in the bush. Major Bonnin had no hesitation in saying that in the great majority of cases of backache those present would encounter, X-ray examination was unnecessary.

Major Bonnin went on to discuss briefly the commoner conditions that would be met with. Referring to fibrositis, he said that the onset, especially that of the first attack, was often sudden and might follow unaccustomed exercise, exposure to cold or some minor strain. The pain was usually referred to a wide area of the back. Pain, as a rule, was caused by movements which stretched rather than compressed the painful area, and if the lesion was in muscle or tendon, pain was greater on active movement—for example, on rising from the bent position. The chief diagnostic feature was the discovery of one or more localized points of acute tenderness to deep pressure. Injection of those points with a local anæsthetic agent both proved the diagnosis and was often dramatically effective therapy. That aspect would be discussed later. In many of these cases a septic focus was present, and recurrent attacks were likely if that was not dealt with.

Referring to sacro-iliac strain, Major Bonnin said that the term was used for want of a better; the condition was not common, but not rare. Pain in the sacro-iliac joint might be due to tearing of some fibres of the massive supporting liagaments, to fibrositic changes in those ligaments or to slight arthritic changes in the joint itself, and it seemed likely that in many cases all three conditions might be present to some degree. The onset might be sudden with no obvious cause as in cases of fibrositis; but commonly it followed some strain, such as a sudden lift with the weight on one side of the body, as in cranking a car, or a lift with hands above the head as in raising a window. The patient complained of pain, maximal in intensity, over the dorsum of the joint and often with a wide area of radiation, and there might be an acutely painful "catch" on his straightening up from the bent position. The diagnosis was easy. There was very severe pain over the affected joint when it was wrenched as described, and there was local tenderness over the dorsum of the joint. If the trouble followed a recent injury, the treatment consisted in rest, followed as soon as possible by graduated exercises. In a condition of longer standing a manipulation under anæsthesia, followed by a short period of rest, and again graduated exercises, usually resulted in a cure. Such patients usually required about three weeks before they could return to duty, and consequently they should be evacuated from a regimental aid post.

Major Bonnin went on to discuss lumbo-sacral strain. He said that that term also was a poor one; but it was used with a similar significance to the term "sacro-iliac strain". By it, he meant pain in the region of the lumbo-sacral joints due to mild trauma (especially recurrent or chronic), or to deep fibrositic or periarthritic or mild arthritic changes in or about the lumbo-sacral joints. This made the term a fairly wide one; but on the other hand, it was just about as accurate as the diagnosis in many cases, although often a shrewd guess as to the exact nature of the lesion could be made. Pain in this region was often associated with faulty posture, especially lordosis. If deformity was present (for example, a sharp lordosis at the lumbo-sacral junction), if pain was severe, and especially if it was referred along the distribution of the sciatic nerve, or if the lesion did not respond to the methods of treatment that would be described, the patient should be evacuated for X-ray examination, for congenital anomalies in that region were common and various, and it was the site for rupture of an intervertebral disk. Treatment would be discussed later; but it could be said at once that the key to treatment was to correct the postural deformity by suitable exercises. In the adult, full correction was, of course, often impossible; but that was not necessary for relief of symptoms. It was necessary only to obtain sufficient correction to take the strain off the aching ligaments. The injection of a local anæsthetic agent might again make possible an accurate diagnosis and provide a cure. Three patients treated at the hospital within the last month had been completely relieved by injection of the interspinous ligament between the fifth lumbar and first sacral vertebra combined with suitable exercise.

Major Bonnin next referred to sciatica and the ruptured intervertebral disk. He said that by sciatica he meant a pain down the back of the thigh to the leg and usually foot associated with deep tenderness in the hamstrings. The ætiology in many cases was still uncertain; but the position was slowly becoming clarified. The opinion was becoming unanimous that true neuritis apart from general polyneuritis was an extremely rare cause of such pain. Perineural fibrosis might explain some cases, but evidence of fibrositis in other places was needed, and all other likely causes had to be excluded before that diagnosis could be entertained. It was becoming generally accepted that the commonest single cause of sciatic pain was the rupture of an intervertebral disk. Describing the structure of the disks, Major Bonnin said that each had a plate of hyaline cartilage above and below separating it from the cancellous bone of the vertebral body. Peripherally, there was a fibrocartilaginous ring, and the centre was the soft *nucleus pulposus*, the remains of the old notochord. It was possible that what happened in the case of rupture of a disk was the following sequence of events: following trauma, of the nature of an extra crush to the already compressed edge of a disk, the cartilaginous ring was cracked or damaged; rapid degeneration followed, with softening and fragmentation of the fibrocartilage, until a few days later the body weight exploded the compressed soft central nucleus out through the degenerating cartilage ring. If, as often happened, this rupture was posterior in position, the extruded mass then impinged on the posterior longitudinal ligament; this was stronger centrally than laterally, so that the protruding mass usually went to one or other side and came up against an emerging nerve root, which it compressed, stretched or irritated. In more than 95% of cases, the rupture affected the disk between the fourth and fifth lumbar or fifth lumbar and first sacral vertebræ, and usually it was the fifth lumbar nerve root that suffered. In a typical case, the patient gave the history of some injury, such as lifting an over-heavy weight into the back of a lorry, or leaving a train and missing his step. He suffered a sudden pain in the lower part of his back and might "feel something go". This pain soon wore off, and he might continue working. A few days later the pain recurred and extended down the back of his thigh and usually the outer side of his leg and foot. Characteristically, the pain was not continuous, but varied from time to time. Examination revealed nothing very much. The patient stood with a slight list away from the side of the lesion. Bending toward the side of the lesion or hyperextending the back caused pain, and if the patient was made to maintain this posture for a short while, the pain in the leg increased and became unbearable. The hamstrings might be tender, and pain was caused by flexing the hip

with the knee extended; the ankle jerks might be diminished or lost and sensation to light touch on the outer side of the foot dulled. Treatment in the first instance was by rest, with perhaps extension or the application of a plaster cast, and in the last resort, operation. Other causes of sciatica were many and included osteoarthritis of the spine and malignant disease. Sciatica was thus a symptom and not a disease. Patients suffering from sciatica should be evacuated to a general hospital for investigation.

Referring to osteoarthritis, Major Bonnin said that he would not discuss its classification. The onset was gradual, with recurring attacks of backache, and later pain might be referred to areas supplied by segmental nerves. The diagnosis was suggested by relatively painless limitation of movement, active and passive, in all directions over the affected segments of the spine. Diagnosis must be confirmed by X rays, and treatment was little more than recategorization. Septic foci should be eliminated.

Major Bonnin said that the conditions which he had briefly described included the vast majority of the causes of low backache pain that those present would encounter, and he was not concerned with rarities. Having taken the history and carried out the routine examination described, they had to make the decision whether to retain the patient or to evacuate him to hospital. By far the greater number of the cases that they would encounter were the "fibrositis-strain and mild arthritis" type, and such patients could and should be retained and treated effectively in the regimental aid post. Most of the patients with lesions requiring their evacuation had already been mentioned. The methods of treatment available were both simple and effective. Exercise was the most valuable of all forms of treatment, and it was used at some stage in the treatment of practically all patients with backache. It should be used for all patients retained at the regimental aid post. Apart from physical benefit, it forced the patient's cooperation in his recovery and prevented the invalid outlook. Virtually the only contra-indication was acute pain or a recent strain. The usual physical training back exercises as taught in army schools of physical instruction would serve for most patients; but when the posture was bad, special exercises in addition were necessary. Lordosis was the commonest postural fault. The patient should be made to lie on the floor and flatten his back against it and then to carry out leg exercises. He should stand with his back flattened against a wall or post and carry out arm and leg exercises in that position, and then stand away from the wall, maintaining the posture, and repeat the exercises; he should be encouraged to maintain the posture during the day.

Major Bonnin went on to say that deep local massage was a rapidly effective form of treatment in some cases. It was indicated in early fibrositis, and it was effective in many cases of late fibrositis. Describing the technique, Major Bonnin said that first the tender point should be accurately located; then, with thumbs or with the fingers reinforced by the other hand, the medical officer should proceed to knead it deeply and as firmly as the patient could stand. The area under the thumb would gradually become less sensitive, and the pressure should be increased constantly. The treatment should continue for several minutes and be followed by exercises. Early fibrositis would often be cured by one treatment of this nature. The standard regimental aid post orderly's rubbing of the skin with liniment was pleasant for the patient, but not effective therapeutically, and it was mentioned only to be condemned.

Major Bonnin then referred to treatment by the injection of a local anæsthetic agent. He said that injection into a painful spot was not only invaluable for diagnosis, but often dramatically effective in treatment. If the injection of procaine into a tender area abolished the symptoms, then it was a fair inference that the cause of the trouble had been located. The indication for an injection was the presence of a localized, painful area of at least some days' duration. Describing the technique, Major Bonnin said that one should first locate the painful area accurately by deep palpation. If one was using procaine, a wheal should be raised on the skin over this point. The hypodermic needle should be pushed through the deep fascia and the muscle below infiltrated. It had been said that the needle should be pushed on until the tender area was encountered; but Major Bonnin had usually been unable to locate it with the needle point. He generally began injecting as soon as the needle was felt to pass the fascia, and he pushed it on for a distance of half an inch to one or two inches depending on the depth of the muscle. It was unnecessary to use more than about 0·5 or 1·0 cubic centimetre. The needle was then withdrawn and the injected area heavily massaged. All tenderness should have gone. If it had not, the needle should be reinserted over any remaining tender spot and a little more anæsthetic solution injected; the spot should be massaged again. When the tenderness had gone, the patient should be put through the movements and manipulations which had previously caused pain. If the injection had been successful, they should now be entirely painless. If they were not, then another tender spot might be present which had been missed. It should be sought and dealt with in similar fashion. When the injection was completed, the patient should be put through his full range of movements, and perhaps should be given passive stretching and active exercises. Exercise should be continued during the next week or so. This after-treatment was most important. If "Novocain" or procaine was used, usually complete relief lasted for from one to eight hours, and then the symptoms were worse. During the next few days improvement was said to follow. Major Bonnin said that he had not been very pleased with the results of procaine injection, except for diagnosis. He had usually used it in the first instance as being entirely harmless, and then four or five days later, if the immediate result was good and the late result was not satisfactory, he had injected the same area with "Proctocaine". The technique was the same, but a skin wheal must not be raised; 0·5 cubic centimetre was usually quite enough anæsthetic solution if the tender point had been accurately located. The immediate result was just as dramatic as when procaine was used; the next day symptoms often recurred to some extent, and thereafter relief was almost complete. Again, it was important to follow the injections with exercise for a week or more.

Major Bonnin went on to say that manipulation without anæsthesia and passive stretching were indicated in the type of case associated with mild, chronic or recurrent strain, fibrositis or arthritis, in which no well-localized tender spots could be found. It was a particularly good form of treatment for the "bludger". It should not be used when a recent injury had occurred, when the patient was suffering much pain, or when any deformity or suspicion of any serious disease was present. The procedure was simple; the patient was subjected to the manœuvres described in connexion with the clinical examination, only considerably more force should be used. Unless a hold like the Boston crab was applied, it was almost impossible to do harm without anæsthesia. The manipulation had to be followed by exercises. It was good to apply a more gentle, passive stretching, using the same movements before the exercises each day. It might often be necessary to repeat the manipulation. Rest should be used for these patients as a Scotsman spent money, only when absolutely necessary and then as little as possible. It was indicated only in such conditions as disk lesions, for a short time after a strain, in hyperacute lumbago, and in general when very severe pain was present—in other words, for the patients who were to be evacuated. As soon as possible gradually increasing exercises should be begun.

Major Bonnin summarized his remarks by saying that although the causes of backache were legion, and although exact diagnosis was in some cases a matter of extreme difficulty, yet by the application of an ordinary routine clinical examination, an accurate diagnosis could be made in a large proportion of cases and a reasonably accurate tentative diagnosis in as many more. Moreover, in quite primitive surroundings, simple and effective treatment could be applied. He again stressed the importance of setting aside fifteen minutes for the investigation for a patient with backache, of taking a careful history, and of stripping the patient completely naked. With a little practice, the examination soon became a routine procedure. In general, the patients who would be evacuated were suffering from acute conditions associated with severe pain—for example, hyperacute lumbago and sacro-iliac strain and sciatica, and conditions giving rise to suspicion of some serious lesion. If circumstances were favourable, the opportunity should be taken to evacuate the patient to hospital for removal of septic foci. Methods of treatment at the disposal of medical officers comprised exercises, deep local massage, injection of local anæsthetic solution, manipulation and passive stretching, and (last and least important) rest; when all was considered, little more that was really effective could be done in a well-equipped massage department. An intelligent regimental aid post orderly could soon be trained to carry out most of this treatment, and physical training instructors were available in most units. Major Bonnin again reminded those present that all these cases of backache were potential cases of neurosis, and he again stressed the importance of dealing with them early and decisively. Incomplete examination, followed by doubt, hesitation, and finally "passing the buck", was the best way to start or perpetuate

a neurosis. These patients should not be put on "no duty" unless it was really necessary; if they were not fit for full duty, they should be given such light duties as they could do to occupy their minds. In conclusion, Major Bonnin expressed his thanks to Captain R. S. Lawson, whose suggestions, he said, were responsible for a large part of what might be found of use in his discussion on low backache.

Correspondence.

THE FUTURE OF MEDICAL PRACTICE.

SIR: Every medical man should carefully peruse Sir Raphael Cilento's brilliant advocacy of a salaried service as published in today's journal. It alone solves the problem of economic security for the doctor, not so much for us older men, but for the younger men at present in the Services and for our sons in the coming generation. It gives the medical man the opportunity for recreation and for study and ends the twenty-four hour a day accessibility which no other profession tolerates. It solves the problem of a better service for the patient, who will have a group of men, all collaborating, without any fear of competition, for his welfare, and it ends the haunting fear of a man where illness can cost him his life's savings. Dr. Watson's advocacy of a fee-for-service at 1s. 6d. per visit will, I am sure, cause much mirth among those with knowledge of small country towns, where six and not sixty consultations would be the daily average. He also indirectly advocates the salaried service, when he says that "many patients have been comforted in their anxieties by a little simple natural psychology". How many hours are wasted by doctors, for the sake of their practices, humouring neurotics instead of "coming to the point" and giving a truthful diagnosis?

Yours, etc.,

A. HAMPDEN MURPHY.

Kingsgrove,
New South Wales,
April 10, 1943.

SIR: Your issue of April 10, 1943, contains a very interesting letter on this subject by Dr. Watson, which calls for comment both on the destructive and constructive side.

Firstly, may I suggest that of all the discussion that has appeared in your columns, the large majority has come from the pens of the senior members of our profession, and again mostly from those in practice in the cities. Very little active discussion seems to have been entered either by the younger ones or by those in smaller country centres. It is conceded that these senior members have a far greater wealth of valuable experience of the conditions of practice, but surely the younger ones like myself, who for some reason are still in practice and debarred from active military service, could take a more active interest in these far-reaching discussions, which are likely to affect us far more than our seniors.

Likewise, much more of the attention seems to have been paid to the suburban areas, and relatively little to the less populated country towns, where conditions of practice are so radically different.

Dr. Watson has produced so far the most drastic condemnation that has appeared of the salaried service, and in principle I am in agreement that a salaried service would not work for Australia. However, his criticism in one respect rather exceeds the bounds, in assuming that under a salaried service we would all naturally become a bunch of cold-hearted, unsympathetic, icy automatons, all too anxious to be burdened with as few nuisances of patients as possible, as long as we collect our salary. No, Dr. Watson, you have a very poor view of your colleagues in an honourable profession. I most sincerely hope that when this new order comes, as come it apparently must, we will continue to give our patients the same treatment under any scheme and uphold our ethical standards against any degrading influence.

I agree that the fee-for-service system seems probably the most workable one, with the two possible objections that, firstly, it would involve a considerable amount of book-keeping, and secondly, that it could be open to abuses. The remaining choice of a capitation scheme would mean less clerical work, and the present type of lodge practice seems to work quite satisfactorily, at least in the country, but I have the feeling that I would rather work under a modification of the present system of private practice.

Finally, as regards rate of payment, Dr. Watson suggests two shillings each for sixty consultations daily and thirty on Saturdays. Now might I respectfully ask whether Dr. Watson has any conception of the conditions of practice in smaller country towns, where a daily average of 25 consultations may be doing fairly well. Doctors in small towns are very necessary, but I think that they would either have to be paid at a slightly higher rate to make a living, or else the more remote areas would have to be provided for by a separate and adequate salary. Besides, although I am well aware that many do it regularly, what doctor, particularly in a large country practice, could do justice to sixty consultations daily, in addition to hospital rounds, operations, confinements *et cetera* without descending to the veterinary level and jeopardizing his own health?

Yours, etc.,

J. H. PRIESTLEY.

Canowindra,
New South Wales,
April 13, 1943.

SIR: In your issue of today's date there is a letter from Dr. A. Leslie Watson in which occurs an extraordinary passage. While no general practitioner in Australia would take the inference seriously, I fear lest other eyes than those of general practitioners, perchance even non-professional ones, should read the letter and their owners be misled.

Speaking of his proposal for improving the standard of general practice, Dr. Watson writes, "Say 60 consultations daily, 30 on Saturdays—". And in case any should complain of the idle life led by the general practitioner in this particular Utopia we are airily told that the 60 is "without extras" or private patients.

On a number of occasions I have questioned fellow general practitioners as to the number of patients they could see per hour in their surgeries without seriously reducing the standard of their work. The almost invariable answer has been three. Note that this does not include little extras like anæsthetics, operations, travelling time, correspondence, and reading either articles or two-column letters in THE MEDICAL JOURNAL OF AUSTRALIA. Really, my dear Watson . . . !

Yours, etc.,

T. DUDLEY HAGGER.

Bingara,
New South Wales,
April 15, 1943.

THE PRACTICAL RESULTS OF IMMUNIZATION AGAINST DIPHTHERIA.

SIR: Dr. Ralph Weaver, in your number of April 10, published some interesting figures showing the splendid results of his intensive immunization campaign in the city of Brisbane. Some time ago a similar campaign was initiated in Toronto, the capital city of the province of Ontario, Canada, with the result that there were no cases of diphtheria in 1940 and 1941. I have no information yet about 1942. The population of Toronto (approximately 670,000) is about double that of Brisbane. Previously diphtheria was rife there.

Another fine example of the practical benefits of applied preventive medicine in Ontario is the abolition of bovine tuberculosis in human beings in that province. Pasteurization of milk, which had been compulsory in the larger towns for some years, was made obligatory for the whole province in 1939. Ontario has an area one-third larger than that of New South Wales, and a population of 3,757,000.

Yours, etc.,

HILARY ROCHE.

Austin Hospital,
Heidelberg,
Melbourne.
April 17, 1943.

MEDICAL PLANNING.

SIR: I have recently read with great interest the scheme put forward by Dr. Davis, of Tamworth, and appreciate his thoughtful study. Arising out of my reading certain points present themselves as worthy of stress.

1. What I, and probably many others, most desire is the elimination of the question of whether the patient can afford this or that. All writers who express satisfaction

with the patient paying "such fees as he can afford" entirely overlook a big human factor and appear to have no idea of the fundamental injustices involved.

2. An inclusive fee as suggested by Dr. Davis would get over that. However, I was disappointed to see compensation cases regarded as an extra: if these are somehow included in a flat rate we would not have men taking on these jobs only. The insurance premiums could probably be adjusted so that the patient injured in his own backyard could be covered equally with the patient injured at work.

This elimination of the financial stimulus to attend cases of injury requires to be extended to include hospital honoraries as much as the general practitioners.

3. I was pleased also to see the specialist placed on the same level as the general practitioner, for I see no fundamental reason why they should be regarded as of more value to the community than the all-rounder; provided only that the general practitioner has reasonable opportunity to study his work and has facilities to care for cases proper to his sphere.

Extra time commonly spent by specialists in study abroad is not peculiar to them and, on the other hand, the harder conditions of work and longer hours in general practice require consideration. Hospital appointments should, of course, be paid appointments and this would relieve the early lean years of specialism.

Yours, etc.,

143, Macquarie Street, Sydney, April 18, 1943. C. C. McKELLAR.

Australian Medical Board Proceedings.

QUEENSLAND.

THE undermentioned has been registered, pursuant to the provisions of *The Medical Acts*, 1939-1940, of Queensland, as specialist in medicine:

Parry, Trevor Alexander, on active service.

The undermentioned has been registered, pursuant to the provisions of *The Medical Acts*, 1939-1940, of Queensland, as specialist in hygiene and tropical diseases:

Croll, Darcy Graham, on active service.

Obituary.

JAMES CHARLES HUGHES.

WE regret to announce the death of Dr. James Charles Hughes, which occurred on April 30, 1943, at North Sydney, New South Wales.

Books Received.

"Manual of Oxygen Therapy Techniques Including Carbon Dioxide, Helium and Water Vapor", by Albert H. Andrews, junior, M.D.; 1943. Chicago: The Year Book Publishers, Incorporated. 7¼" × 4¾", pp. 191, with illustrations. Price: $1.75, post paid.

"The 1942 Year Book of General Surgery", edited by Evarts A. Graham, A.B., M.D. Chicago: The Year Book Publishers. 7¼" × 5", pp. 848. Price: $3.00, post paid.

"Out of Working Hours: Medical Psychology on Special Occasions", by Henry Yellowlees, O.B.E., M.D., F.R.F.P.S. (Glasgow), F.R.C.P. (Edinburgh), F.R.C.P. (London), D.P.M.; 1943. London: J. and A. Churchill, Limited. 7½" × 4¾", pp. 160. Price: 8s. 6d.

"Hygiene for Nurses", by John Guy, M.D., D.P.H., F.R.F.P. and S. (Glasgow), F.R.C.P. (Edinburgh), and G. J. I. Linklater, O.B.E., M.D., D.P.H., D.T.M. and H., M.R.C.P. (Edinburgh); Sixth Edition; 1943. Edinburgh: E. and S. Livingstone. 7½" × 5", pp. 248, with illustrations. Price: 5s. net, postage 5d.

"First Aid in Civil Defence", Civil Defence Handbook Number 3, issued under the authority of the Minister for Home Security; 1942. Sydney: Government Printer. 7" × 4¾", pp. 157, with 74 illustrations.

[1] "A Guide to the Preservation of Life at Sea after Shipwreck". Medical Research Council: Committee on the Care of Shipwrecked Personnel: War Memorandum Number 8; 1943. London: His Majesty's Stationery Office. 9¼" × 6", pp. 21, with 2 illustrations. Price: 4d. net.

Diary for the Month.

MAY 11.—Tasmanian Branch, B.M.A.: Branch.
MAY 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAY 14.—Queensland Branch, B.M.A.: Council.
MAY 18.—New South Wales Branch, B.M.A.: Ethics Committee.
MAY 19.—Western Australian Branch, B.M.A.: Branch.
MAY 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
MAY 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 26.—Victorian Branch, B.M.A.: Council.
MAY 27.—New South Wales Branch, B.M.A.: Branch.
MAY 27.—South Australian Branch, B.M.A.: Branch.
MAY 28.—Queensland Branch, B.M.A.: Council.
JUNE 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 2.—Victorian Branch, B.M.A.: Branch.
JUNE 2.—Western Australian Branch, B.M.A.: Council.
JUNE 3.—New South Wales Branch, B.M.A.: Special Groups Committee.
JUNE 3.—South Australian Branch, B.M.A.: Council.
JUNE 4.—Queensland Branch, B.M.A.: Branch (Joseph Bancroft Memorial Lecture).
JUNE 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 8.—Tasmanian Branch, B.M.A.: Branch.
JUNE 11.—Queensland Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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